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Ischemic heart disease

Anatomy of the coronary arteries

	The left Coronary artery		Right Coronary
Origin	Left sinus of Valsalva		Right sinus of Valsalva
Site	Left atrioventricular groove		Right A-V groove
Branches	Anterior descending anterior inter-V groove ----- apex -- ---- turns backwards	The circumflex artery left A-V groove	Marginal a + posterior descending a
Anastomosis	posterior descending a	Right coronary	circumflex artery
Supply	Anterior surface	marginal branches Lateral surface	Inferior surface

Pattern of coronary supply

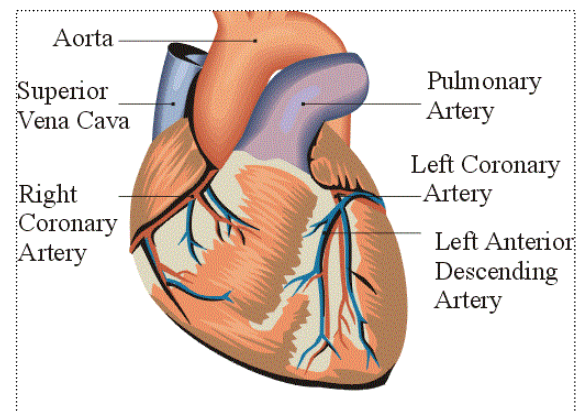
1- Balanced circulation:

Left coronary:- supplies L atrium, L ventricle + *anterior part of the inter-V septum.*

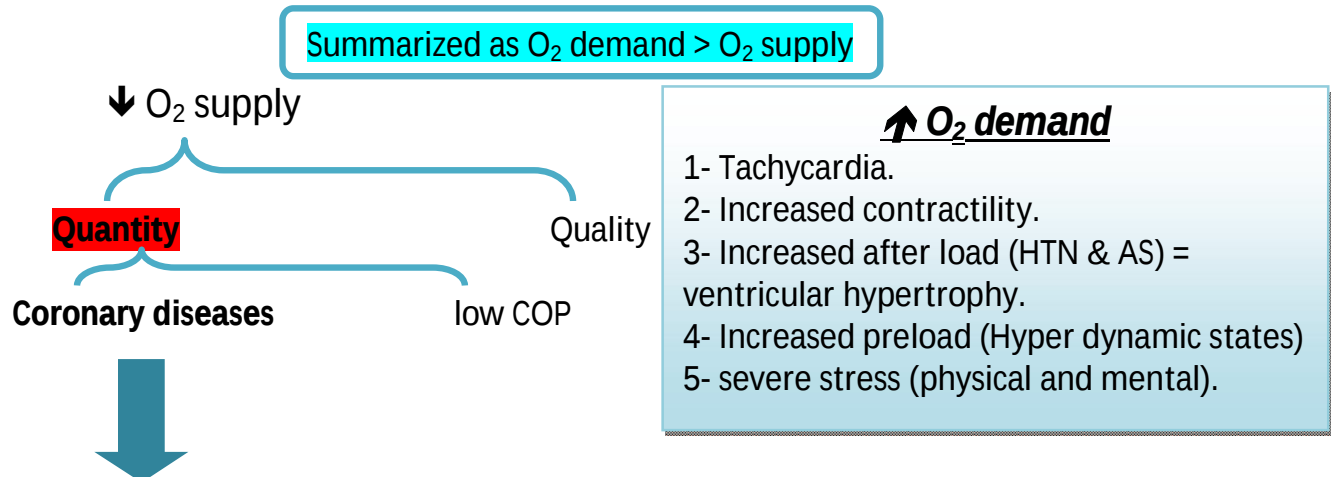
Right coronary:- supplies R atrium, R ventricle + *posterior part of the inter-V septum.*

2- Right dominance:- Right coronary supplies also the posterior part of the L. ventricle.

3- Left dominance:- Left coronary supplies also the posterior part of the septum & the posterior wall of the right ventricle.



Aetiology of ischemic heart disease



Decreased oxygen supply

A. Decreased quantity of coronary blood:

1. **Coronary artery disease:** in ability of coronary to maintain sufficient blood flow in spite of good cardiac output.

Intima	• Atherosclerosis (the most common cause). • Vasculitis e.g. PAN
Osteum	• Coronary osteal stenosis e.g. syphilis, calcification. • Congenital anomalies.
Musculosa	• Dissection. • Spasm.
Leumin	• Embolism. • Coronary thrombosis in hyper-coagulability (polycythaemia).
Others	• Microcirculation vasoconstriction. • Spontaneous coronary dissection.

2. **Low cardiac output states:** e.g. valvular stenosis especially AS, Pulmonary hypertension.

B. Decreased quality of coronary blood

- Hypoxia.
- Severe anemia.

Risk factors of atherosclerosis

A- Non-modifiable

- Age ----- (Major)
- Male sex ----- (Major)
- Family History:- ----- (Major)

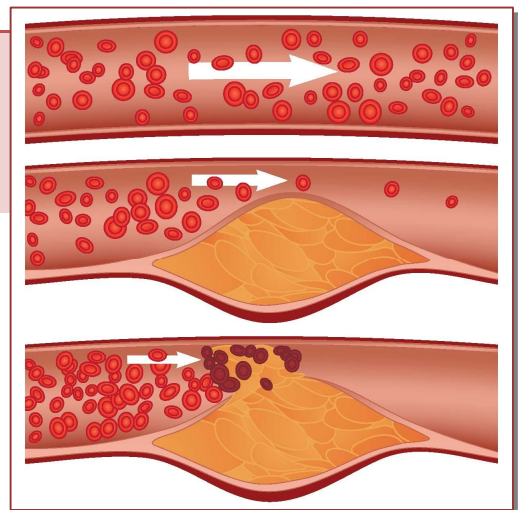
Close relatives of complicated atherosclerosis (IHDs or stroke) + Genetic (as familial hypercholesterolemia)

B- Modifiable:-

- Systemic hypertension---- (Major).
- Cigarette smoking ----- (Major).
- Obesity.

C- Partially Modifiable

- Diabetes Mellitus ----- (Major).
- Hypercholesterolemia ---- (Major).
- Dyslipidaemia ----- (Major)
 - High LDL and / or VLDL
 - Low HDL "protective "
 - LDL/HDL ratio > 3:1



D- Ps

- **Physical inactivity.**
- **Psychic stress.**
- **Personality (mentally active ambitious individuals).**

Others:-

Elevated serum levels of triglycerides + homocysteine + uric acid + fibrinogen
+ Elevated serum insulin levels (Insulin resistance)
Elevated C-reactive protein Vitamin B₆ deficiency
Hypercoagulability Postmenopausal estrogen deficiency
High intake of saturated fat + carbohydrate

Metabolic syndrome:- 1- DM:- hyperinsulinemia " insulin resistance" 2- abdominal obesity 3- decreased HDL cholesterol levels 4- Hypertension

Preventive factors:- A high serum HDL cholesterol level, physical activity, estrogen, and moderate alcohol intake (1 – 2 drinks /day)

Presentation of ischemic heart disease "clinical pictures"

- A. **Asymptomatic**: silent myocardial ischemia.
- B. **Symptomatic** :
1. Chest pain:
 - Angina pectoris: stable angina, unstable angina, variant angina.
 - Acute myocardial infarction.
 2. Other presentation: (with or without chest pain) e.g.
 - Heart failure.
 - Arrhythmias.
 - Fatigue.
 - Syncope.
 - Sudden death.

Angina pectoris

Definition clinical term used to describe **chest pain** resulting from **brief** myocardial ischemia (oxygen demand > oxygen supply).

Pathophysiology

Chemical and mechanical stimulation of sensory afferent nerve endings in the coronary vessels and myocardium. These nerve fibers extend from the T1 – T4

Chemical:- anaerobic metabolism of myocardium accumulates **lactate & pyruvate**.

Adenosine may be the main chemical mediator of anginal pain.

Mechanical :- Heart rate, myocardial inotropic state and myocardial wall tension are the major determinants of myocardial metabolic activity and myocardial oxygen demand.

Anginal pain

- Onset → acute.
- Course → intermittent.
- Duration → 30 sec. – 30 min.
- Effect of treatment → respond.

Don't forget 11 points:

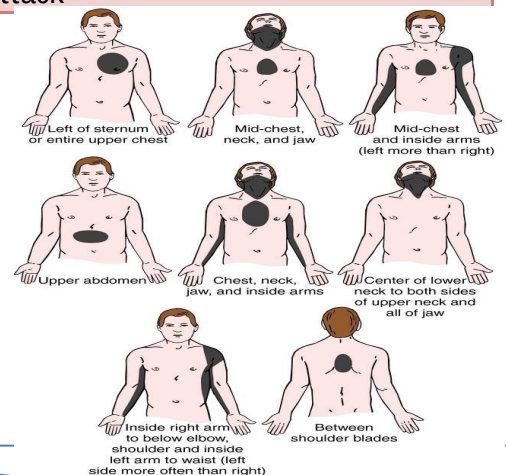
- Onset / Course / Duration.
- Association.
- What ↑ and what ↓.
- Effect of TTT.
- Date of last attack.
- +3 (site, radiation and character)

Site	Retrosternal or precordial
Radiation	Left shoulder, medial aspect of left arm, forearm, hand, neck, lower jaw, epigastrium, right shoulder and back.
Character	Constricting, compressing or in the form of discomfort, tightness oppression or sense of suffocation. (never to be stiching)
Precipitating	Muscular exercise, sexual intercourse Emotional stress الضغط , heavy smoking, heavy meals, cold atmosphere (hypothermia) or high altitudes..
Relief ing	Rest, sublingual nitrates.
Duration	Few minutes, usually less than 20 min, but may be prolonged.
Association	Dyspnea, palpitation, sense of fear of death (angor animi), sweating, dizziness, fainting, nausea, vomiting and eructation at the end of the attack

Physical signs

There may be no abnormality

- **General examination:** during the attack may reveal:
pallor, sweating, rise of HR & ABP.
- **Cardiac examination:** may reveal:-
 - ✓ Weak heart sounds especially S₁.
 - ✓ Reversed splitting of S₂.
 - ✓ S₄.
 - ✓ Murmur of papillary muscle dysfunction.



Types of angina

Stable	The pain is relatively constant as regards severity, precipitating factors and relief.
Unstable	• Angina of recent onset (within 2 months). • Or, angina at rest. • Or, angina with increased duration, frequency or severity (crescendo angina)
Variant	(prezmetral angina, angina inversa, vasospastic angina) • Angina at rest & not precipitated by decreased oxygen demand. • It is usually associated with (S – T segment elevation). • It is most probably due to coronary spasm. • Coronary arteriography may be normal.
Other types	<u>Angina decubitus</u>: Angina that is precipitated by lying down & improved by sitting. <u>Nocturnal angina</u>: Angina that awakens the patient at night. <u>Angina of Lewis</u>: Occurring in severe aortic incompetence e.g. syphilitic characterized by being nocturnal, prolonged & associated with excessive sweating.

Any other forms of angina classified as unstable angina.

Investigations

A. For diagnosis of angina

1. ECG:

Resting	In between the attacks : it may be normal <i>During pain : may show 1 of 4:-</i> : S-T segment:- depression (horizontal or down sloping) : T wave: inverted, flat or tall & peaked : Arrhythmias:- any type : Heart block.	
Exercise	May show 1 of 4:- : ST segment depression : Ventricular arrhythmias. Typical angina pain during the test or hypotension	
<i>Contraindications:-</i> Acute attacks - Severe hypertension - Orthopedic problems - Severe AS - Congestive heart failure.		
Ambulatory ECG (holter):- for undiagnosed angina or variant angina		

N.B. :- S-T segment elevation in variant angina.

2. Echocardiography & Doppler ultrasound

May show regional wall motion abnormalities during rest or induced by exercise.

3. Radionuclide studies: using Thallium 201

Ischaemia appears as a filling defect (**cold spot**) at exercise & disappears after rest.

4. Coronary arteriography:

Indications:

- Intractable angina not responding to medical treatment
- Unstable angina
- Post infarction angina
- Undiagnosed chest pain
- Before coronary revascularization.

Benefits:

It demonstrates the number, site, extend & severity of obstructive lesions.

B. Detection of risk factors of atherosclerosis

- Blood glucose level
- Plasma lipid profile: cholesterol, LDL, HDL and triglycerides.

Differential diagnosis:

- Differentiation from other causes of chest pain especially
 - ✓ Cardiac causes :- e.g. myocardial infarction & Mitral valve prolapsed
 - ✓ Gastrointestinal e.g. oesophageal spasm, gastro-oesophageal reflux
 - ✓ Musculoskeletal disorders, e.g. cervical spine disease.
- Determination of the type of angina: stable, unstable or variant.

Treatment of Angina Pectoris (4)

- 1- General
- 2- Medical
- 3- Revascularization
- 4- TTT of Acute attack

A- General Measures:-

1. Control of risk factors of atherosclerosis:- e.g.
 - Smoking - Hypertension - Hyperlipidaemia - Obesity
2. Avoidance of precipitating factors:- e.g.
 - Severe exertion - Heavy meals - Emotional stress, tranquilizers may be helpful.
3. Control of aggravating factors:- e.g.
 - Anaemia - Hypoxia - Thyrotoxicosis
4. Exercise Program:- should be regular, gradual, dynamic (e.g. walking) and should be finished after pain or tachycardia.

Effect of regular exercise:-

- Decrease:- heart rate, BP & coagulability (decrease platelet aggregation & activate fibrinolytic)
- Increase:- collateral coronary blood flow & effort tolerance (Orbelli)

B. Medical treatment

	Mechanism	Drugs & doses	Side effects
Nitrate	1. Reduce O ₂ demand:- - Venodilator (Preload) - Arteriodilator (After load) 2. Dilatation of coronary.	A. Short-acting:- <u>Nitroglycerin</u> (on C- GMP) ▪ Sub-ling :- 0.3-0.6 mg ▪ Oral:- 2.5mg/6-12 hours. ▪ Spray or Patch B. Long-acting: <u>Isosorbide dinirate</u> :- 5mg SL during pain - 10-40 mg t.d.s. <u>Isosorbide mononitrate</u> : 20 mg bid.	• Headache, flushing, • Nausea, • Postural hypotension, • Dizziness, syncope. • Sudden withdrawal may precipitate angina.
B-blockers	Reduce O ₂ demand by decreasing:- • Heart rate • Blood pressure • Myocardial contractility, (No coronary effect)	Non-selective: e.g. • <u>Propranolol</u> : 40-80mg/6-12 hours. • <u>Nadolol</u> Selective: e.g. • <u>Atenolol</u> (Tenormin): 50-100 mg/d • <u>Metoprolol</u> (hyophilic cross BBB) • <u>Esmolol</u> is a short-acting IV	(As nitrate)+ • <u>Bronchospasm</u> • <u>Bradycardia</u> & HB • <u>Precipitation</u> of HF • <u>Peripheral V. disease</u> • Masking hypoglycemic manifestations in DM • <u>Depression</u> (in old) • <u>Impotence</u>
Ca-blocker	As 1 + 2	Verapamil :- 80-120 mg/8 h orally Diltiazem :- 30-90mg/8 h orally Amiodipin : 5mg/d orally	(As nitrate)+ • Bradycardia. • heart block.
Nifedipine : 10-20 mg/8 h orally (Not used) tachycardia aggravating angina			

B-blocker may be useful in patients with unstable angina.

Ca. blockers: may be useful in *no response to B-blockers, or when contraindicated*
 – In patients with **Prinzmetal** angina with or without nitrates.

Anti-platelets:

1. Aspirin: - (anti cyclooxygenase ----- suppression of thromboxan A₂)

- Antiplatelet effect can last as long as 7d.
- Dose:- 81-325mg

Side effects

- Hypersensitivity – liver damage; hypoprothrombinemia; vitamin – K- deficiency; bleeding disorders and asthma.
 - Reye syndrome (children (<16 y) with flu)
2. Clopidogrel: - Selectively inhibits ADP binding to platelet receptor-75mg PO qd
 3. Ticlopidine (Ticlid: 250 mg/12 h)
 4. Picvklamole (Persantin): 75mg tds

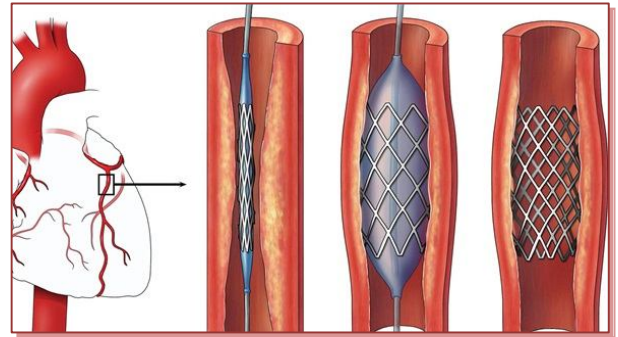
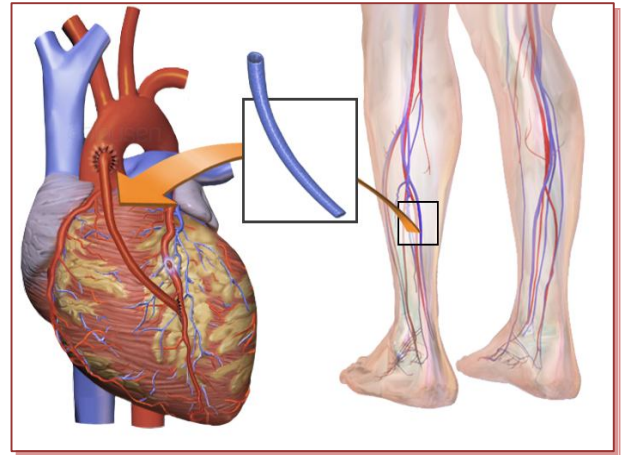
Heparin: May be given in (unstable angina) not responding to other drugs.

C- Coronary Revascularization:Indications

- Intractable angina (failure of medical)
- Three vessel disease
- Post infarction ischaemia

Procedures:

- **Surgical**: - Coronary artery bypass graft **(CABG)** using internal mammary artery or saphenous vein graft.
- **Interventional** :- Percutaneous transluminal coronary angioplasty **(PTCA)**: for
 1. Balloon angioplasty for thrombectomy.
 2. Coronary stenting, laser angioplasty.

**D- TTT of acute attack:**

- Complete rest
- Nitroglycerine (0.5 mg) or isosorbide dinitrate (5 mg) sublingually & repeated up to 3 times successively with interval of 3 minutes
- If the patient is not relieved after the use of 2-3 tablets, the patient should be immediately transferred to hospital & evaluated for the possibility of myocardial infarction

Myocardial infarction

Definition :- Complete **coronary obstruction** leads to an **ischaemic necrosis** of a part of the cardiac muscle

Aetiology

Causes of coronary ischaemic heart disease (see above) but usually it occurs as a result of **thrombosis on top of coronary atherosclerosis**.

The site of infarction depends on the occluded artery:-

- Occlusion of anterior descending artery: causes anterior infarction.
- Occlusion of circumflex artery: causes lateral infarction
- Occlusion of right coronary artery: causes inferior infarction.

Clinical picture

(Symptoms + complications + Signs)

- Patients with myocardial infarction may present with chest pain (described as angina pain with differences), complications or both.
- The condition, however, may be asymptomatic.

A. Symptoms

1. Chest Pain

- It is the main presenting symptom.
- It differs from anginal pain in the following features:
 - ✓ It is more severe and usually crushing or squeezing in nature.
 - ✓ It is more prolonged.
 - ✓ It may occur without precipitating factors or after severe mental stress.
 - ✓ It is not relieved by rest or sublingual nitrates.
 - ✓ It may be associated with many complications.

2. The pain may be absent (painless infarction) Silent myocardial ischaemia

Definition: lack of symptoms in spite of sure ECG or nuclear evidences of ischaemia.

Caused by:

- a. Autonomic neuropathy especially in diabetics.
- b. Anesthesia, sedation or impaired consciousness.
- c. Infarction occurring after cardiac transplantation.
- d. In old age.

Type I: - Totally asymptomatic patients.
Type II: - Patients with previous MI.
Type III: - Patients with chronic angina.

B. Complications(5, 5)

Early (5)	Late (5)
1- Shock:- Cardiac - neurogenic 2- H.F:- LVF - RVF 3- Arrhythmia:- Common - dangerous 4- Heart: End/Myoc/Peri-cardium 5- Sudden death	1- Post inf. S. 2- Aneurysm 3- Thromboembolism 4- Frozen shoulder 5- Post inf. angina

The patient may present by one or more of the following complications:

Early complications

1. Shock

	Cardiogenic shock	Neurogenic shock
Cause	Failure of pump (massive MI)	Severe pain leads to vagal stimulation
C/P	Irritability, cold sweats, pallor peripheral cyanosis.	Peripheral vasodilatation &
Pulse BP	Rapid weak Pulse - Hypotension - oliguria. $\pm$ APO	Bradycardia Hypotension
Others	P. congestion or APO	Nausea & vomiting
prognosis	poor	Good (response to analgesic)

2. Heart failure

	Left ventricular failure	Right ventricular failure
Incidence	The most common	Secondary to LVF - Rare after RV- MI.
Chronic	low CO & P. congestion.	low CO & S. congestion.
Acute	APO (no cardiomegaly)	
prognosis	Bad	Good

3. Arrhythmia

Common	Dangerous
Any type of arrhythmia or heart block can occur	
- Extrasystole	- Ventricular tachycardia -V. fibrillation - Asystole
- Partial HB	- Complete HB.

4. Heart**a. Endocardium:** (Thrombo-embolism)

Mural thrombosis: in the LV --- may lead to systemic embolism (e.g. cerebral).

b. Pericardial effusion:

- Dry pericarditis may occur.
- Haemorrhagic effusion or haemopericardium may develop
- Post-infarction syndrome "Dressler's syndrome" See below"

c. **Myocardial rupture**; May lead to

Direction	Leads to	Manifested by
Outward	Haemo-pericardium	Cardiac tamponade may lead to death.
Inward	Rupture of septum	Acquired VSD
Midway	Rupture of papillary muscle	Acute mitral Regurge --- Acute HF

5. Sudden death:

Due to ventricular fibrillation, ventricular asystole or rupture of the heart.

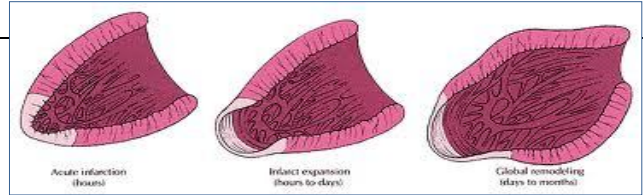
Late complications

1. Post-infarction syndrome "Dressler's syndrome":

- It is pleura-pericarditis developing 2-6 weeks after infarction.
- It is an immune reaction due to release of antigens from necrotic cardiac muscle.

2. Myocardial aneurysm:-

Presented with	Examined with
- Refractory heart failure. - Recurrent embolism. - Recurrent arrhythmias. - Rupture ---- haemopericardium.	- Cardiac exam.:- may show double apex - weak S1 - ECG :- persistent elevation of the S-T segment - Echocardiography or screen:- shows paradoxical P.



3. Thromboembolism:

- Mural thrombosis**: in the LV --- may lead to systemic embolism (e.g. cerebral).
- DVT**: due to prolonged recumbency & may cause pulmonary embolism.

4. Frozen shoulder: It is stiffness & pain of the left shoulder due to reflex arteriolar VC leading to ischaemia & fibrous or due to limitation of movement.

5. Post-infarction angina & reinfarction:

Due to affection of other coronary arteries

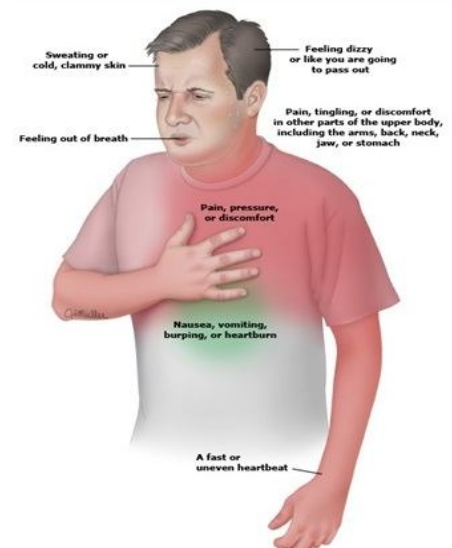
C. General signs

General appearance:

The patient may be pale, sweaty, irritable, dyspneic

Pulse:

	Explained by
Tachycardia:	heart failure & cardiogenic shock.
Bradycardia:	to excessive vagal stimulation or heart block.
Irregular:	may be present in cases of arrhythmias.
Small Volume	due to heart failure or shock.



Blood pressure:

- **Hypertension** may occur due to sympathetic stimulation.
- **Hypotension** in shock
- **Decapitated B.P.:** Marked drop of systolic + slight drop in the diastolic.

Body temperature:

Fever: may be as moderate form - starting in the second day - lasting for few days.

Neck vein:

Congestive pulsating: if there is RVF or haemopericardium.

Cardiac Signs**Heart sound:-**

- Weak heart sounds especially S1
- Paradoxical splitting of S2.

Additional sound:-

- S3: May be due to diminished myocardial compliance, or
- S1: due to flabby myocardium, especially with pericardial effusion.

Murmurs:

- **MR:** pansystolic or late systolic murmur due to papillary muscle rupture
- **VSD:** pansystolic murmur due to septal rupture

Pericardial rub: May be present in cases associated with pericarditis.

Differential diagnosis

Other causes of acute chest pain (see symptomatology) e.g.

- Unstable angina.
- Massive pulmonary embolism.
- Dissecting aortic aneurysm.
- Dry pericarditis & acute pleurisy.

Investigations (5+5)

5 Labs (biochemical)	5 Images
Enzymes CBC ESR Bl. Glucose TG & cholesterol	ECG Echo RNS X ray Catheter

1- ECG:

Pathological Q wave	Necrosis	(Old MI)
Raised coved S-T segment	Injury pattern	(Recent MI)
Inverted T wave	Ischaemia	

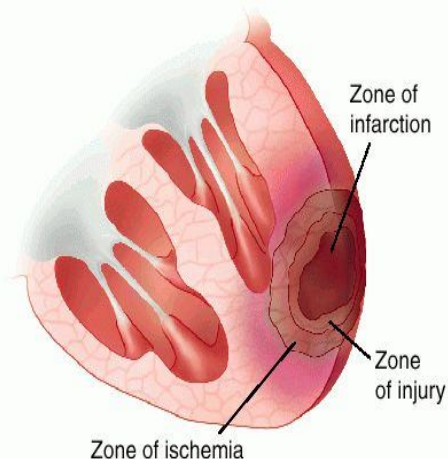
- T wave may be tall & peaked
- Different types of arrhythmias & HB may occur.

The site of infarction could be detected from the ECG leads:-

- Inferior infarction: in leads II, III & aVF.
- Lateral infarction: in leads avL, V5 & V6.
- Anteroseptal infarction : in leads VI to V4
- Extensive anterior infarction : in leads avL & VI – V6

N.B.

- **Right ventricular** – RV4, RV5
- **Posterior wall** – R/S ratio > 1 in V1 and V2 ; T-wave changes (i.e, upright) in V1, V8, and V9.

**2- Echocardiography & Doppler ultrasound**

- The infarcted area may show regional wall motion abnormalities during rest.
- Complications may be detected e.g. mural thrombi, mitral incompetence.

3- Radionuclide studies

Techetium-99m:- taken up by the myocardium in proportion to the blood flow.

- Indicated in patients with atypical presentations or unclear ECGs.
- The infarcted area appears as **hot spot**.

Thallium scan: the infarcted area appears as a cold spot.

4- X-ray:- in acute HF usually no chamber enlargement could be detected

5- Coronary angiography:- (as in angina)

- For demonstration and assessment of the condition of other coronary arteries.
- It is useful for demonstration of the site & size of occluded vessel.

B- Biochemical

a. **Enzymatic changes:**

	Normal level	Elevation	Peak	Ended
Creatine phosphokinase (CK)	40-180 units/L	4-8 h	24-48 h	3-5 d
SGOT	8-40 units/L	6-8 h	24-48 h	4-6 d
Scrum lactic dehydrogenase (LDH)	150-300 units/L	12-24h,1	48-72 h	7-14 d

Elevation of these enzymes is not specific for myocardial infarction.

The most important is the CK-especially the cardiac isoenzyme CK MB.

b. **Myoglobin:**

- This is a very sensitive early marker - but it is not specific.

The myoglobin / carbonic anhydrase III ratio increases specificity (Myocardial cells do not release carbonic anhydrase).

c. **Troponin 1:** contractile protein - released only when myo-necrosis occurs.

- Its sensitivity is superior to that of the CK-MB.
- Troponin 1 is detectable 3-6 hours after an AMI- remains elevated for 14 days.

d. **Complete blood count:**

- Leukocytosis may be within several hours - It peaks in 2 - 4 days - normal after 1 week.

e. **Erythrocyte sedimentation rate (ESR):**

- Rises within 3 days - may remain elevated for several weeks.

Treatment of the myocardial infarction "5, 5, 5"

i. Pre hospital

1. Rapid transfer to hospital is a must (Time lost is life lost).
2. Oxygen inhalation.
3. For heart block → **A- Atropine** 0.5 – 1 mg **IV**.
4. Analgesics for pain → **M- Morphine** 5 – 10 mg **IV**.
5. For ventricular arrhythmias → **L- Lidocaine** 50 – 100 mg **IV**.

ii. Hospital care

1. General

- a. Admission to CCU (coronary care unit) continuous hemodynamic monitoring & ECG.
- b. Oxygen inhalation.
- c. Diet: light frequent meals & avoid constipation.
- d. Sedative: Diazepam.
- e. Aspirin: **an essential element** (325 mg initial dose then 75 mg daily-oral).
- f. ACE inhibitor: oral therapy e.g. Lisinopril 5 mg on day 1 & 2, then 10 mg daily.

- *ACE inhibitors are vasodilator that reduce myocardial demand and Ventricular remodeling (changes in size, shape, structure and physiology of the heart after injury to the myocardium).*
- *In DM:- should be in mild hyperglycemia as hypoglycemia cause tachycardia.*

2. Relieving of chest pain

- a. Morphine (4 mg IV every 5 to 10 minutes as needed).
- b. Nitroglycerine.
- c. Beta blockers. } To relieve pain of post infarction angina.

3. Thrombolytic therapy "time is muscle"

- The earlier that thrombolytic therapy is given after the onset of chest pain, the greater the benefit (*thrombolytic therapy is beneficial up to 6 hour but may be given for up to 12 hours*)

Drugs:

- **Streptokinase:** 1.5 million units IV over 60 min. may cause allergy.
 - **Urokinase.**
 - **Alteplase:** "tissue plasminogen activator – tPA".
- Anti coagulant "Heparin" & anti platelets "Aspirin" are given with and after thrombolytic therapy to reduce the risk of re-occlusion.

Contraindications "the major risk is bleeding"

- Bleeding disorders.
- Major surgery within past 2 weeks.
- Recent cerebral hemorrhage within past 12 months.
- Active internal bleeding e.g. peptic ulcer.
- Severe hypertension.
- Diabetic retinopathy with recent bleed.
- **Aortic dissection.**
- **Pericarditis.**

4. Angioplasty Percutaneous Transluminal Coronary Angioplasty (PTCA):

- Introduction of balloon or stent to dilate the stenotic artery (Balloon-tipped catheter).
- More effective than thrombolytic therapy (fewer complication, short hospitalization).

5. Treatment of early complication e.g.

- Acute heart failure.
- Arrhythmia.
- Cardiogenic shock.

iii. After discharge

1. Reassurance & rehabilitation "gradual return to normal activity".
2. Treatment of post infarction angina.
3. Treatment of precipitating factors e.g. hyperlipidemia.
4. Treatment of late complications e.g. myocardial aneurysm: aneurysmectomy.
5. Aspirin & Beta blockers "decrease the risk of post infarction angina & reinfarction".

Acute coronary syndrome "ACS"

Are myocardial infarction and unstable angina, as there are similar pathophysiologic mechanisms.

1. Unstable angina.
2. AMI:
 - STEMI: confirmed by cardiac markers "enzymes".
 - NSTEMI: diagnosed by cardiac markers "enzymes".

Heart failure

Definition

Inability of the heart to **maintain cardiac output** sufficient to metabolic **requirements** of the body ***in spite of adequate venous return.***

Classification of HF

1. Left-sided heart failure, right-sided heart failure or biventricular.
2. Acute & chronic heart failure.
3. Systolic & diastolic failure.
4. Aetiological classification e.g. Ischaemic heart failure.
5. According to cardiac output:

Low C.O.P. failure	High C.O.P failure
- C.O.P. less than 5L/min - most of HF cases except those with hyperdynamic circulation	- C.O.P. high but net insufficient for tissue needs. - This occurs in hyperdynamic circulation - Treated by treatment of the cause.

Aetiology of heart failure

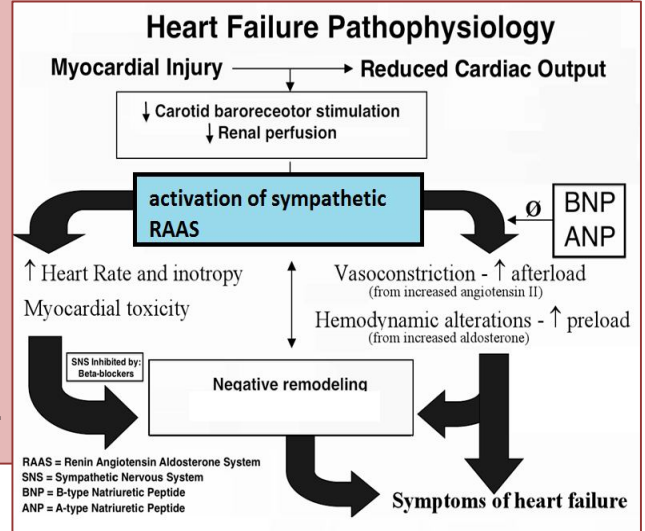
	Left Atrium	Right Atrium
	* M.S * Left atrial myxoma	* Pulmonary hypertension - T.S . * Right atrial myxoma
	Left ventricle	Right ventricle
Increased Volume overload	* Mitral incompetence - Aortic incompetence. * VSD & PDA. * Hyperdynamic circulation.	* Tricuspid incompetence. * VSD & PDA. * Hyperdynamic circulation.
Increased Tension overload	* Systemic hypertension (HTN). * Aortic stenosis. * Coarctation of aorta.	* Pulmonary hypertension. : Mitral stenosis. : LVF. : Chest dis. (Cor-pulmonale). * Massive pulmonary embolism. * Pulmonary stenosis.
Decreased contractility (Myocardial disease)	* Ischemic heart disease especially MI. * Cardiomyopathy. * Myocarditis.	

Causes of biventricular failure:

- * + right ventricular failure on top of left ventricular failure (**congestive HF**).

Precipitating factors ((3I + 3P + 2A))

- **I**nfections: as infective endocarditis, Lung infection.
- Myocardial **I**nfarction.
- **I**atrogenic: discontinuation of anti failure therapy excess salt, IV fluids, steroid, -Ve inotropic (B blocker)
- **P**hysical and emotional stress.
- **P**regnancy & labour.
- **P**ulmonary embolism.
- **A**rrhythmia e.g. marked tachycardia or bradycardia.
- **A**nemia, thyrotoxicosis ,, hyperdynamic circulation.

**Clinical pictures****Left-sided heart failure****A) Forward manifestations (low COP) "after"**

- 1- forward:- Low COP
- 2- Backward:- P. Congestion
- 3- CVS:- General + Local

Symptoms	Signs
– Easy fatigability & LL intermittent claudication. – Dizziness, blurring of vision & may be syncope. – Anginal pain in severe cases. – Oliguria in severe cases	– Pallor, coldness & peripheral cyanosis. – Weak pulse. – Low systolic pressure.

B) Backward manifestations (pulmonary congestion) "before"

Symptoms	Signs
– Dyspnea: – <i>Exertional dyspnea --- dyspnea at rest</i> – <i>Orthopnea</i> – <i>Paroxysmal nocturnal dyspnea ---- PND</i>. – Cough - Hemoptysis - Recurrent chest infections	– Bilateral basal crepitations (BBC). – Pleural effusion (hydrothorax). – In severe cases: acute pulmonary edema (APO)

C) Cardiovascular signs**1. General signs:**

- **Tachycardia**: due to sympathetic stimulation
 but may be absent:- in cases associated with HB, digitalis, B. Blockers.
- **Weak pulse**.
- **Pulsus alternans**: alternating strong & weak beat with equidistance
- Systolic pressure: **low**

2. Local signs:**Precordial examination**

- Signs of left ventricular enlargement.

Auscultation

- Ventricular (protodiastolic) gallop "at apex".
- Murmur of functional mitral incompetence due to left ventricular dilatation.
- Features of the cause may be detected.

Right-sided heart failure

- 1- forward:- Low COP
- 2- Backward:- S. Congestion
- 3- CVS:- General + Local

A) **Forward manifestations (low COP)** "after" See above.

B) **Backward manifestations (systemic congestion)** "before"

Symptoms

- Pain in the right hypochondrium & epigastrium.
- Dyspepsia, anorexia, nausea, vomiting & may be malabsorption in severe cases.

Signs

- Enlarged tender liver & may be cardiac cirrhosis in chronic failure cases.
- Congested pulsating neck veins.
- Ascites. - Pleural effusion (hydrothorax).
- Oedema of the lower limbs.

D) Cardiovascular signs**3. General signs:**

- **Tachycardia:** due to sympathetic stimulation
but may be absent:- in cases associated with HB, digitalis, B. Blockers.
- **Weak pulse.**
- Systolic pressure: **low**

4. Local signs:**Precordial examination**

- Signs of Right ventricular enlargement.

Auscultation

- Ventricular (protodiastolic) gallop "at Tricuspid area".
- Murmur of functional Tricuspid incompetence due to Right ventricular dilatation.
- Features of the cause may be detected.

American Heart Association Stages of Heart Failure

Stage	Description
A: High risk for developing HF	Hypertension, diabetes mellitus, CAD, +Ve family history
B: Asymptomatic HF	Previous disorders:- MI, LV dysfunction, valvular heart disease
C: Symptomatic HF	Structural heart disease - SOB - fatigue - impaired exercise tolerance
D: Refractory end-stage HF	Marked symptoms at rest despite maximal medical therapy

Summary

	LVF	RVF
Forward manifestations	Low COP	
	Symptoms • Syncope. • anginal pain and oliguria. • Intermittent claudication.	Signs • Pallor, coldness & peripheral cyanosis. • Weak pulse. • Low systolic pressure.
Backward manifestations	Pulmonary congestion Symptoms • Dyspnea, orthopnea & PND. • Cough. • Haemoptysis. • Recurrent chest infection. Signs • Bilateral basal crepitations (BBC). • Pleural effusion. • APO.	Systemic congestion Symptoms • Right hypochondrial pain. • Dyspepsia. • L.L. edema. Signs • Enlarged tender liver. • Congested neck veins. • Ascites. • Pleural effusion. • L.L. edema.
General signs	• Tachycardia "except???" • Weak pulse. • Pulsus alternans. • Low systolic pressure. • Pleural effusion (hydrothorax).	• Tachycardia "except?" • Weak pulse. • Low systolic pressure. • Pleural effusion (hydrothorax).
Local cardiovascular signs	• Signs of LV enlargement. Auscultation • S3 gallop (at apex). • Murmur of functional MR due to LV dilatation. Features of the cause	• Signs of RV enlargement. Auscultation • S3 gallop (at tricuspid area). • Murmur of functional TR due to RV dilatation. Features of the cause

Investigations

A. X-ray	– Chamber enlargement. – Pulmonary vascular (left sided failure). – Pleural effusion (Right or left HF). – Calcified valve.
B. ECG	– Chamber enlargement (mainly hypertrophy). – Aetiology (ischaemic)
C. Echo & Doppler	– Cause (e.g. diagnosis of valve lesion or congenital defects). – Site. – Effect (chamber enlargement). – Function (ejection fraction).
D. Cardiac Catheter	– As Echo and doppler

Transesophageal echocardiography :- Is particularly useful in patients who are on mechanical ventilation or morbidly obese and in patients whose transthoracic echo was suboptimal.

B-type natriuretic peptide (V. important) **Diagnostic**

- BNP:- amino acids polypeptide originated from the ventricles.

Radionuclide multiple gated scan

Very reliable imaging technique for determining global heart function.

CBC, KFTs and electrolytes:- Mild anemia can worsen prognosis

Treatment of heart failure

Guidelines for treatment of heart failure:

- 1. Treatment of the underlying etiology.**
- 2. Treatment of the precipitating factors.**
- 3. Treatment specific to the syndrome of HF:**
 - **Decreasing the cardiac load:**
 - a. Rest: physical and emotional.
 - b. Reduction of preload:
 - Diet control, especially salt restriction.
 - Diuretics.
 - Vasodilators.
 - c. Reduction of after load:
 - Vasodilators.
 - **Decreasing the myocardial damage:**
 - a. Beta blockers.
 - **Increasing the myocardial contractility:**
 - a. Digitalis.
 - b. Other inotropic agents:
 - o Sympathomimetic **amines**: dop**amine** & dobut**amine**.
 - o Phosphodiesterase inhibitors: am**rinone** & mil**rinone**.
 - **Resynchronizing the ventricles: "CRT"**
 - a. In patients with cardiomyopathy who have the problem of :
Intraventricular conduction delay or bundle branch block
 - **Symptomatic treatment:**
 - a. For hypoxemia: oxygen administration.
 - b. For cardiac dyspnea: aminophylline administration.
- 4. Treatment of the refractory heart failure.**
- 5. Treatment of acute pulmonary edema.**

1. Rest

- Reduce metabolic requirements

- Semi-sitting or sitting position:- to decrease venous return & improve respiratory muscles.

Complications of prolonged recumbency:

Peripheral	Central	Locomotor system
- DVT. - Bed sores.	- P embolism. - Hypostatic pneumonia. - Constipation.	- Retention of urine. - Depression.
		- Muscle wasting. - Osteoporosis. - Joint stiffness.

2. Sedative

To relieve anxiety (Diazepam or Morphine; the best of APO)

3. Diet

- Salt restriction:** To reduce salt & water retention decreasing blood volume (reduction of preload) (reduction of circulatory congestion & edema).
- Fluid restriction:** in severe & resistant cases of heart failure.
- The meals:** light of carbohydrate mainly and fractionated.
- Caloric restriction:-** Reduction of body weight in obese patients.

4. Diuretics

They promote loss of salt & water decreasing blood volume (reduction of preload).

Diuretics classified into

Diuretic	Site and mechanism	Side effects
Loop Diuretic • Frusemide (lasix) 40-160 mg (O/IV) • Bumetanide • Ethacrynic acid: 50 – 200 mg (O/IV)	Thick ascending loop of Henle inhibition of Na- K & KCL co-transporter	<u>Hypo:</u> kalaemia, natraemia, calcaemia , dehydration, alkalosis. <u>Hyper:</u> glycemia, lipidemia, uricemia. <u>Others:</u> blood dyscrasia, rashes, ototoxicity, GITD, interstitial nephritis
Thiazide • Hydrochlorothiazide 25 – 100 mg /d Or • Chlorothalidone 25 – 100 mg /d Or • Indapamide *	DCT Inhibition of NaCl cotransport Vasodilator	<u>Hypo:</u> kalaemia, natraemia, dehydration, alkalosis <u>Hyper:</u> glycemia, lipidemia, uricaemia, calcaemia <u>Others:</u> Thrombocytopenia, rashes, pancreatitis, impotence.
K sparing • Spironolactone 50 – 200 mg • Triamterene 100 – 300 mg Amiloride 5 – 10 mg /d Or	Aldosterone antagonists Inhibition of Na conductance	Gynaecomastia + Hyperkalaemia Acidosis

*Works even if GFR is below 50 ml/min.

Other diuretics:

- Osmotic diuretics
- Carbonic anhydrase inhibitors e.g. acetazolamide
- Drugs with a diuretic action e.g. dopamine, digitalis
- Mercurial diuretics (not use now)

Causes of diuretic resistance:

- Delayed absorption of the diuretic.
- Reduced secretion of the diuretic into the tubular lumen (its site of action).
- Compensatory retention of sodium after the effective period of the diuretic.
- Hypertrophy and hyperplasia of epithelial cells of the distal convoluted tubule.

5. Vasodilators

Aim reduction of preload (venodilatation) and after load (arteriolar dilatation).

Arteriolar	Venous	Both
Reduce afterload	Reduce preload	Reduce both
: Hydralazine. : Diazoxide.	: Nitrates	: ACEIs. : Na nitroprusside.

1. **Angiotensin-converting enzyme inhibitors (ACEI):** e.g.

- Caplopril : 6.25 - 25mg /8h orally.
- Enalapril.

2. **Ang II receptor Blockers (ARBs):**

- Losartan
- Candesartan

ARBs:- Highly recommended alternatives to ACE inhibitors in patients who cannot tolerate ACE inhibitors because of adverse effects, most notably coughing.

3. **Direct vasodilators:** e.g.

- Nitroglycerine: 0.5 - 5 mg
- Nitroprusside: 0.5 - 10 ug /kg/min IV Infusion
- Alpha-blockers: e.g. Prazocin

4. **Calcium channel- blockers :**e.g.

- Nifedipine.

They are rarely used in heart failure due to their negative inotropic effect.

5. **Hydralazine:-** was the first oral balanced (afterload and preload reduction) vasodilator and was popular before the availability of ACE inhibitors.6. **Beta-adrenergic blocking agents (metoprolol, carvedilol)**

- Improve symptoms and exercise tolerance,
- Reduce ABP so improve LV ejection fraction (**decrease mortality rates**)
(*ischemic and idiopathic cardiomyopathy*).

Was not used before

6. Inotropic Agents

1. Digitalis glycosides.
2. Sympathomimetics:
 - **Dopamine:** 3-10 ug /kg /min IV infusion - **Dobutamin:** 5-10 ug /kg /min IV infusion
3. Sympathomimetics agent:- Amrinone, milrinone.
4. Glucagon.
5. Calcium.

Digitalis

- Increases myocardial **contractility** (i.e. positive inotropic effect) leading to :
 - Increased SV & CO. - Increased systolic BP.
 - Increased renal blood flow & urine output.
 - Decreased venous pressure. - Decreased size of the heart.
- Increases **excitability** of atria & ventricles (may be complicated by arrhythmias).
- Decreases the **automaticity** of SAN (direct & vagal++) slowing the rapid heart.
- Decreases the **conductivity** of AVN (may be complicated by heart block).
- Vagal stimulation.
- ECC changes
 - Digitalis effect: sagging depression of ST segment
 - Flat or inverted T wave.

Digitalis toxicity: different types of arrhythmias & heart block may occur

Mechanism:

- Inhibition of Na-K/ ATPase increasing intracellular Na which increases intracellular calcium.
- Calcium ions promotes sliding of actin & myosin increasing the force of contraction.

Indication:

- Heart failure
- Atria fibrillation, atrial flutter & paroxysmal supra ventricular tachycardia.

Contraindications:-

Absolute	Relative
- Digitalis toxicity. - Ventricular tachycardia (++) excitability)	- Partial Heart block (decrease conductivity). - Nodal rhythm (suppression of S-A node).

Preparations:-

- Oral:** (one tablet equals one international unit)
 - Digoxin: excreted mainly by the kidney (tab = 0.25mg).
 - Digitoxin: metabolized mainly by the liver (tab = -0.1 mg).
- Intravenous preparations:**
 - Digoxin (amp. Contains 0.5 mg).
 - Cedilanid. - Ouabin.

Administration

- Oral:- Previously**

A loading or digitalizing dose of 12-15 units over:

- One day (rapid digitalization)
- 3 Days (moderate digitalization)
- 7 Days (slow digitalization)

This is followed by a maintenance dose of 1-3 units/day.

OR It is given as follows:

- A dose of 2 units daily without loading.
- An optimum therapeutic level is reached after about 5 days.
- This is followed by a maintenance dose of 0.5 - 2 units/day.

✓ **Now :-** Dose of 0.5 - 1 units given daily with stoppage of 1-2 days weekly.

- **Intravenous**

Indications:- Acute HF, AF, A. flutter & PSVT.

Dose:- 0.5 mg / 4h (0.5-1 mg in 5% glucose over 30 min) (not exceed 1.5 mg)

Then maintenance oral dose of 0.5-2 nits/day (not exceed 2.5 units/d)

Digitalis drugs interaction:- Amiodarone , propafenone, quinidine, verapamil, nifedipine, diltiazem, levothyroxine, cyclosporine, flecainide, disopyramide, omeprazole, tetracycline, and erythromycin.

Digitalis toxicity:-

Predisposing factors: - Hypokalemia. - Hypercalcemia - Renal failure. - Liver failure.

Clinical picture:-

1. **GIT:-** Anorexia (the first symptom) - nausea & vomiting.
2. **CVS:-** Different types of arrhythmias & heart block
- Extrasystole (pulsus bigeminus or trigeminus) - Ventricular tachycardia.
3. **CNS:-** Blurring of vision & colored vision (yellow or green).
- Headache, psychosis & neuralgia.

With prolonged use Gynaecomastia may occur.

Treatment:

1. **Stoppage** of digitalis.
2. Correction of **predisposing** factors.
 - a. Correction of hypokalaemia if present by:
 - Reduction of the dose or stoppage of loop diuretics
 - Potassium chloride orally with monitoring of serum K (by ECG):
 - b. Calcium-chelating agents e.g. EDTA are rarely used now.
3. **Symptomatic** treatment:
 - For arrhythmias: antiarrhythmic drugs e.g. propranolol & lidocaine.
 - For heart block: atropine IV
 - For vomiting: antiemetic : Metoclopramide.
4. **Immunotherapy:** Digoxin immune Fab Immunoglobulin fragment with specific and high affinity for both digoxin and digitoxin molecules.
5. **Binding** agents: to reduce absorption e.g. Activated charcoal.
6. **Cholestyramine:** Used to break enterohepatic circulation.

Dopamine

- (sympathomimetics agent) 3-10 ug / kg / min IV infusion = **dose dependent.**

Dose	Stimulate	Effect
Low (0.53 mcg / kg / min)	Renal vascular beds	Diuresis (Nephrogenic)
Moderate (3 – 10 mcg / kg / min)	Beta receptors	Inotropic (Heart)
High dosage (10 - 20 mcg / kg / min)	Alpha receptors	In shock (Shock)

Dobutamine

- (sympathomimetic agent) 5-10 ug / kg / min IV infusion.
- Beta 1-receptor agonist, with some beta 2- receptor and minimal alpha-receptor.
- Contraindicated in patients with moderate or severe hypotension.

Nor Epinephrine

- for patients with profound hypotension (e.g., systolic blood pressure < 60 mm Hg).

Phosphodiesterase inhibitors

- (milrinone, amrinone) (Oral PDIs have no role).
- Phosphodiesterase inhibitors (PDIs) increase intracellular cAMP, which results in a positive inotropic effect on the myocardium and peripheral vasodilation.

7. Aminophylline

Actions	- Bronchodilator. - Vasodilator. - Positive inotropic. - Mild diuretic
Indications	- Bronchial asthma. - Cardiac dyspnea
Administration	- Intravenous: 5.6 mg/kg slowly IV over 20 min. (average 250 – 500 mg). - Orally or per-rectum.
Side effects	- Hypotension. - Arrhythmias. - Insomnia. - Irritability.

8. Venesection:

Closed or open venesection is rarely used in severe & resistant cases. (not now)

9. Dialysis:

May be indicated in severe cases.

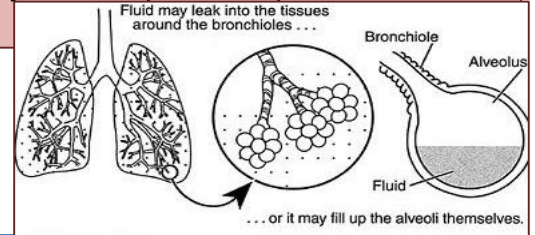
Treatment of diastolic HF

- A. Pericardiectomy** for constrictive pericarditis.
- B. Relief of ventricular systolic overload:**-ACE inhibitors or ARBs.
- C. Anti-ischaemic agents:**
 - B- Blockers - Ca. Blockers - Nitroglycerin
 - (Reducing myocardial ischemia, thus improving ventricular relaxation).
- D. Treatment of the cause:**- Relief of Valvular, Supravalvular, and subvalvular obstruction to ventricular out-flow by operation or balloon valvuloplasty.

Acute pulmonary edema

Definition:- Rapid accumulation of fluid in interstitial & intra-alveolar spaces of the lung.

Cardiogenic pulmonary edema (CPE) is defined as development of pulmonary edema due to increased capillary hydrostatic pressure secondary to elevated pulmonary venous pressure (when LV venous return exceeds LV output).



Etiology

A. Increased pulmonary capillary hydrostatic pressure: (cardiogenic pulmonary edema)

- Acute left-sided heart failure.
- Acute myocardial infarction.
- Severe hypertension.
- Myocarditis.
- Acute mitral or aortic incompetence.
- **Acute exacerbation of chronic LSHF (P congestion)**: e.g. MS with precipitating factors as AF.

B. Increased pulmonary capillary permeability: adult respiratory distress syndrome ARDS (Non-cardiogenic pulmonary edema)

- | | |
|--|--|
| – Shock. | – Septicemia. |
| – Trauma. | – Burns. |
| – DIC. | – Severe pneumonia. |
| – Aspiration of gastric contents. | – Severe hypoalbuminaemia. |
| – Pulmonary lymphatic obstruction. | – High altitudes. |
| – Pancreatitis. | – Terminal renal & liver failure. |
| – Inhalation of gases e.g. chlorine, 100 % oxygen. | – C.N.S. lesions e.g. cerebrovascular strokes & head injuries. |
| – Sudden expansion of a collapsed lung e.g. in rapid aspiration of massive pleural effusion. | |

Clinical pictures

- Severe dyspnea at rest & orthopnea.
- Cough with expectoration of excessive frothy blood-tinged sputum.
- Central cyanosis.
- Marked irritability, sweating & coldness.
- Bilateral crepitations, at first basal then generalized.
- Wheezes may be present.
- Features of the cause e.g. myocardial infarction.

Investigations

- **CBC**: severe anemia or sepsis.
- **Serum electrolytes**: hypokalemia and hypomagnesemia.
- **BUN and Creatinine**.
- **Chest X-ray films**:
 - ✓ Cardiomegaly.
 - ✓ Alveolar edema with pleural effusions (bilateral infiltrates in a butterfly pattern).
 - ✓ Haziness of hilar shadows & loss of sharp definition of pulmonary vasculature.
 - ✓ Thickening of interlobular septa (Kerley B lines).
- **Arterial blood gases**:
 - ✓ Stage 1 and 2: hypoxemia and hypocapnia.
 - ✓ Stage 3: hypoxemia, in severe cases hypercapnia.
- **Pulse oximetry**:
For monitoring the patient's response to supplemental oxygen and other therapies.
- **ECG**:
Not diagnostic.

Treatment of cardiogenic pulmonary oedema (APO)

1. Hospitalization: preferably in ICU & bed rest in sitting position.
2. Morphine: 5 – 10 mg IV.
3. Correction of the cause

Respiratory care	Cardiac care	Circulatory (hypervolaemia)
4. O ₂ inhalation.	6. Digoxin.	8. Frusemide.
5. Aspiration of secretion.	7. Others e.g. Dopamine & Dobutamine.	9. Vasodilators.
10. Aminophylline		

If no response

11. Mechanical ventilation.
 12. Intra-aortic balloon.
 13. Phlebotomy or dialysis.
- **Frusemide**: 20-40 mg IV, repeated every 30 min, according to the response.
 - **Vasodilators**: Nitroglycerine IV or sub, or Na nitroprusside 0.5 - 10 ug / kg / min IV.
 - **Aminophylline**: 5.6 mg/kg slowly IV over 20 min. (average dose : 250-500 mg)
 - **Positive inotropics**: Digoxin may be used especially in cases associated with AF, Other inotropics may be needed e.g. Dopamine & Dobutamine.

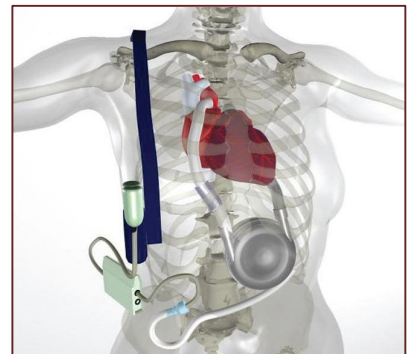
Refractory heart failure

Etiology of refractory heart failure

1. Presence of a mechanical factor hindering the cardiac function: e.g.
 - *Severe valvular disease.*
 - *Pericardial effusion or constrictive pericarditis.*
2. Persistence of the cause: e.g. uncontrolled hypertension.
3. Presence of a precipitating factor: e.g. chest infection.
4. Improper treatment: e.g.
 - *Inadequate rest.*
 - *Inadequate salt restriction.*
 - *Inadequate digitalis.*
5. Terminal cases of heart failure: with advanced myocardial damage, e.g. massive myocardial infarction.

Treatment

1. Removal of the **mechanical factor**: e.g. surgical treatment of valvular disease.
2. Treatment of the **cause**: e.g. proper control of hypertension.
3. Removal of the **precipitating** factor: e.g. proper antibiotics for chest infection.
4. Proper management:
 - *Adequate rest.*
 - *Adequate salt restriction (< 1 gm / day) & fluid restriction in severe cases.*
 - *Adequate digitalis.*
5. For terminal cases of heart failure:
 - *Diuretics*: use combinations e.g. loop diuretics & potassium-sparing diuretics.
 - *Vasodilators*: use combinations of IV vasodilators such as **Na nitroprusside** and a potent IV inotropics such as **Dopamine** or **Dobutamine**.
 - ***Mechanical measures***:
 - ✓ Mechanical removal of fluid using phlebotomy or dialysis.
 - ✓ Mechanical assistance of circulation using intra-aortic balloon counterpulsation: "**Balloon is inflated in diastole & deflated in systole**".
 - *Cardiac transplantation*: in patients below the age of 60.



Rheumatic fever

Definition:

Non suppurative inflammation in immunological base affecting various structures all over the body.

It is preceded by U.R. infection with group A B hemolytic streptococci.

Mechanisms:

1. **Antigenic similarity:** (Cross-reactivity)

Antibodies formed against streptococcal antigens react with human tissue antigens since both are immunologically similar.

2. **Altered antigenicity** (haptene like)

Binding of streptococcal antigens to human tissue antigens, renders them antigenic and stimulates antibody formation against them

The latent period usually 1-4 weeks but in chorea it may be longer (several months).

Predisposing factors:

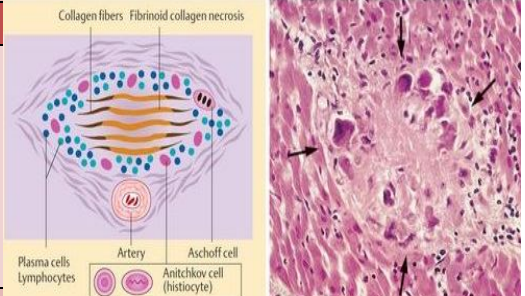
- **Age:** 5-15 years (rare below 4 years & above 26 years).
- **Sex:** equal in both sexes except rheumatic chorea which is more common in females.
- **Familial tendency:** due to both hereditary predisposition & similar environment.
- **Socio-economic factors:** more common in developing countries & low social classes.
- **Geographic distribution:** more common temperate zones
- **Seasonal incidence:** more common in winter.

The inflammatory process:

A. **Site:**

1. *Joints: synovial membrane*
2. *Heart: endocardium, myocardium & pericardium*
3. *CNS: especially basal ganglia.*
4. *Other serous membranes (pleura) + Skin & subcutaneous tissue, Small blood vessels.*

B. **Types of inflammatory reactions**

Exudative lesion	Proliferative: Aschoffe nodule		
Mainly serous membranes. Healing without residual effect.	Affecting mainly the heart. - Central Fibrinoid degeneration - Surrounded by lymphocytes, macrophages, Aschoff giant cells & outer fibroblasts - Healing by fibrosis.		

Clinical picturesA. **Major criteria** "Mnemonic: C.A.N.C.E.R"

- Carditis.
- Arthritis.
- Subcutaneous Nodule.
- Chorea.
- Erythema marginatum.

B. **Minor criteria** "Mnemonic: P.E.A.C.E"

- Prolonged PR interval in the ECG: (**> 0.22 sec due to myocarditis**).
- ESR: elevated.
- Arthralgia: painful joints without swelling.
- CRP (C reactive protein): elevated.
- Elevated temperature "fever".

A. Major criteria**1. Polyarthritis:**

- Number of joint affected :- **polyarticular** (more than 5)
- Distribution: big joints (e.g. knees, ankles, wrists) in a **migratory** character.
- Description: **Arthritis** :- painful, tender, red, hot, swollen, with limitation.
- Drugs: usually subsides spontaneously or dramatically response to **salicylates**.
- Deformity: **no any residual lesion** The joint left (completely free after few days).

jaccoud's arthropathy is a rare late complication

2. Carditis (Pancarditis)

Myocarditis	Endocarditis	Pericarditis
Precordial examination: • Cardiac enlargement • Heart failure:- in severe cases • Different types of arrhythmias & HB may occur. • Disproportionate tachycardia Auscultation • Weak heart Sounds. • Ventricular gallop. • Murmurs of functional mitral & tricuspid incompetence. • Tic- Tac rhythm.	Rheumatic valvulitis:- more in left side (M then A) Carey – Coombs murmur:- relative MS in the acute stage only - explained as edematous cusps. disappears after the acute stage. Rare:- Acute MR or AR during the acute stage due to damage of the cusps. Later on:- fibrosis of the valves may lead to stenosis, incompetence or both.	i. Dry Pericarditis: common. ii. Effusion: may occur. ii. Adhesive pericarditis: Rare. v. Constrictive pericarditis: never.

Presence of pericarditis denotes severe pancarditis.

Arthritis is more common in adults - **Carditis** is more common in children.

Some say

JONES cr **ITERIA**:

Major criteria:

Joint (arthritis).
Obvious (Cardiac).
Nodule (Rheumatic).
Erythema marginatum.
Sydenham chorea.

Minor criteria:

Inflammatory cells (leukocytosis).
Temperature (fever).
ESR/CRP elevated.
Raised PR interval.
Itself (previous Hx of Rheumatic fever).
Arthralgia.

3. Rheumatic Sydenham's Chorea: (See Neurology)**4. Subcutaneous nodules:**

- Size: Small firm nodules (0.5-2 cm in diameter)
- Site: They tend to be symmetrical and occur over bony prominences & tendons.
e.g.: extensor surfaces of elbows, knees & ankles occiput and spinous processes of thoracic & lumbar vertebrae.
- Shape: Small firm nodules not painful, not tender & not adherent to the skin.

They are usually associated with carditis.

**5. Erythema marginatum:**

- Size: variable, the margin progress & the center clears
- Site: These areas appear over the trunk & proximal parts of extremities.
- Shape: areas of erythema non-pruritic, non-painful, not indurated & blanch on pressure.

N.B. They are evanescent & recurrent carditis.

**B. Minor Criteria:**

- Fever.
- Arthralgia.
- History of rheumatic fever or evidence of rheumatic heart disease.
- Acute phase reactants: High ESR - C-reactive protein - PNL leucocytosis.
- Prolonged P-R interval more than 0.22 sec.
- Other manifestations of rheumatic fever: Pallor, sweating, weakness & loss of weight, Epistaxis, Tachycardia, Pleurisy & Pneumonia.

Complications of Rheumatic fever

Early	Late
• Heart failure. • Arrhythmias. • Heart block.	• Rheumatic valvular lesions. • Rheumatic activity. • Rarely: adhesive pericarditis & jaccoud's arthropathy.

Investigations**A. Laboratory investigations**

1. Blood picture
 - Polymorph nuclear leucocytosis.
 - Normocytic anemia.

- 1- acute phase reactants
- 2- streptococcal lab.
- 3- cardiac

2. Erythrocyte sedimentation rate: Markedly elevated (it is not specific for rheumatic fever) but it is one of the minor criteria & also used for follow up.
3. C-Reactive protein: Non-specific

B. Investigations for streptococcal

1. **Anti-Streptolysin O titer (ASOT)** Elevated in 80% of cases.
 - Titers greater than 250 Todd units/ml in adults & 500 Todd units /ml in children are indicative of recent streptococcal infection.
 - A rising titer may be detected early in the course of rheumatic fever.
2. **Streptozyme test:** positive in more than 95% of cases.
3. Throat culture: positive in only few cases.

C. Cardiac investigations:- may show

D. X-ray	– Chamber enlargement. – Pulmonary vascular (left sided failure). - Pleural effusion
E. ECG	– Prolonged P-R interval more than 0.33 sec. – May be inverted T wave. – Different types of arrhythmias & heart block may occur.
F. Echo & Doppler	– Chamber enlargement). – Valve incompetence. – Function (ejection fraction).

Diagnosis of rheumatic fever

Revised Jones Criteria

The diagnosis requires the presence of

- 2 Major criteria, or
- 1 major & 2 minor criteria.

Plus evidence of recent streptococcal infection e.g.

- ✓ Increased anti-streptococcal antibodies e.g. ASOT.
- ✓ Positive throat culture for group A streptococci
- ✓ Rapid streptococcal antigen test.
- ✓ Recent scarlet fever.

Differential diagnosis

1. Causes of fever in a cardiac patient (infective endocarditis).
2. Causes precipitating heart failure.
3. Causes of acute arthritis e.g. juvenile chronic arthropathy, SLE, Acute leukemia, sickle cell anemia, Henoch Schonlein purpura.

Treatment

Prophylactic:

A. Primary prevention:

- Early diagnosis & proper treatment of upper respiratory tract infections.
- Tonsillectomy may be indicated in some patients with recurrent tonsillitis.

B. Secondary prevention:

- Long acting penicillin (Benzathine penicillin G): 1.200.000 units IM Injection monthly until the age of 25 years, for 5 years after the last attack, or for life.
- Erythromycin (for penicillin-allergic patients: 250mg /6hs.

Therapeutic:

1. General

- Complete bed rest until clinical & laboratory findings (e.g. ESR) return to normal.
- Salt restriction if there is heart failure
- Light nutrient diet

2. Symptomatic treatment: e.g.

- Treatment of heart failure.
- Treatment of chorea.

3. Specific

Antibiotics: For eradication of the streptococcal infection

- Benzathine penicillin G 1.200.000-600.000 units IM or - Procaine penicillin: 600.000 units/day for 10 days
- Erythromycin (for penicillin-allergic patients 250 mg/6 hours orally for 10 days.

Anti-inflammatory drugs

	Aspirin	Steroid
Indication:	- Rh. fever without or with mild carditis - Side effects or contraindications for steroids - During & after withdrawal of steroids	- Rheumatic carditis - If salicylates are not effective or not tolerated
Initial dose:	90 - 120 mg / Kg / day	2 mg/kg/day
Then:	<u>After a satisfactory response is obtained</u> (usually after 3 /weeks) The dose is reduced to 60 - 70 mg / kg / day for an additional 6-9 weeks.	<u>After 3 weeks</u> steroids are withdrawn gradually over an additional 3 weeks

(Aspirin should be given during withdrawal of steroids & after discontinuation for 3 weeks)

4- Treatment of complication: e.g.

- Treatment of heart failure.

ENDOCARDITIS

Definition :- infection of the endocardium

A. Infective endocarditis

- According to the causative organism:

- Bacterial endocarditis.

- Non-bacterial endocarditis.

- According to the clinical presentation:

Subacute bacterial endocarditis (SBE)	Acute bacterial endocarditis (ABE)
- insidious onset - affecting diseased hearts - caused by low virulence organism	- a severe disease - acute onset & progressive - affecting diseased or normal hearts - caused by highly virulent organisms

B. Non-infective endocarditis: e.g. rheumatic fever, SLE

Other classification of Infective Endocarditis:

Native Valves	Community-acquired native-valve endocarditis. The commonest form after Mitral-valve prolapsed
Prosthetic Valves	Prosthetic – valve endocarditis 15% of cases of infective endocarditis - may indicate re-replacement
Nosocomial	10% of all cases of endocarditis - hospital acquired
Infective endocarditis in immunocompromised patients	

INFECTIVE ENDOCARDITIS

The development of infective endocarditis requires the combination of two factors.

Bacteraemia. + Underlying cardiac lesion.

A. Bacteraemia:

The causative organisms.

- **Gram-positive cocci:**
 - Strept viridians (the commonest).
 - Strept. Faecalis & Staph. aureus.
- **Gram-negative bacilli:** e.g.
 - Haemophilus, Actinobacillus, Cardiobacterium, Eikenella & Klebsiella.
- **Pseudomonas & Brucella**
- **Other organisms:** Fungi, Rickettsia, Chlamydia
 - **HACEK organisms:** (haemophilus species (Haemophilus parainfluenzae), Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella C.).

Rout	Possible organism
Dental, oral & URT	Strept. Viridans
GIT & genitourinary	Strept. faecalis
Cardiac & catheter	Staph aureus

B. Underlying Cardiac Lesion:

- Valvular lesions:
 - Especially MR, AR & AS.
 - Mitral valve prolapse:- 5-8 times more than normal valve
 - The disease is uncommon in MS because of marked fibrosis & low pressure.
- Congenital anomalies:
 - Especially VSD, PDA & A. coarctation.
 - The disease is rare in ASD due to low pressure gradient between the two atria.
- Prosthetic valves:-

Two factors in cardiac lesions:

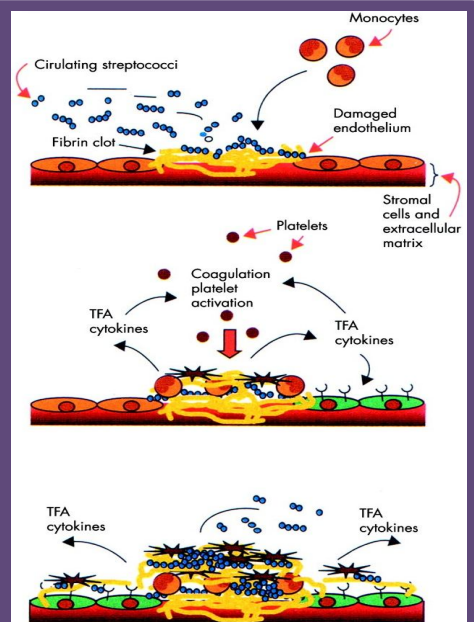
1. High pressure source:
 - It is more common in the LS of the heart.
 - It is uncommon in mitral stenosis, AF & ASD.
2. Narrow orifice: e.g. more common in smaller VSD.

Endocarditis:**A. Vegetation formation:-**

- Over the valvular or mural endocardium.
- Composed of bacteria with blood cells & fibrin.
- May detach leading to embolization.

B. Acute infection may result in Damage & perforation of the cusps or chordate tendinae.

N.B. The (myocardium) may be inflamed

**Clinical picture:-**

1. General toxemia.
2. Cardiac manifestations.
3. Embolization.
4. Immune processes.

Manifestations of the disease include**1- General manifestations:**

- ✓ Fever: mild or moderate & may be continuous, remittent or intermittent.
- ✓ Anorexia, loss of weight & weakness.
- ✓ Marked pallor & toxic face.
- ✓ Pulse Tachycardia is a constant feature or may be absent due to embolization.

Eyes :

- ✓ Sudden blindness: due to embolism of (CRA).
- ✓ Petichiae in the conjunctiva.
- ✓ Roth spots: red patches in the retina.

• **Hands & lower limbs:**

1- Splinter hemorrhages: Longitudinal haemorrhages under the nails due to microemboli & rupture of capillaries.	
2- Osler nodules: Small painful tender intracutaneous nodules occurring in the pulps of the fingers & toes and may be hands & feet (They are due to endothelial hyperplasia of the capillaries on top of vasculitis).	
3- Janeway lesions: Small, painless, blue red lesions appearing on palms & soles.	

4- Pale clubbing.

5- Skin: Pallor, petichiae.

2- Cardiac manifestations:

- Features of the underlying cardiac disease.
- Precipitation of heart failure.
- Alteration of the character of the already present murmurs or development of new murmurs.
- Rupture cusps or chorda tendinae causing a loud musical murmur (**Sea Gull**)

3, 4- Manifestations due to affection of other organs:

Spleen	• Slightly enlarged, soft & tender. • Stitching pain & splenic rub due to embolic splenic infarction.
Kidney	• Acute glomerulonephritis, leading to nephritis, nephritic S. or RF. • Focal embolic glomerulonephritis: leading to microscopic haematuria. • Renal infarction: leading to loin pain & macroscopic haematuria.
C.N.S.	• Cerebral embolism: embolic hemiplegia. • Subarachnoid hemorrhages: mycotic aneurysm at the circle of Willis.
Lung	• Pulmonary infarctions & infections in cases of right-sided endocarditis (rare).
Joints	• Arthralgia or less commonly arthritis may occur.

Complications

1. Heart failure due to: valvular damage, myocarditis.
2. Embolization: (see above).
3. Complications of therapy.
4. Perivalvular extension of infective endocarditis.
5. Prolonged Fever.

Investigations:

A. **Laboratory investigations:**

- **Blood culture:**

- ✓ It is the most important investigation & is positive in about 85% of cases.
- ✓ 6 samples of blood are taken.
- ✓ Samples are cultured under aerobic & anaerobic conditions.
- ✓ The results are looked for after 3 days & up to 60 days.

Bone marrow culture may be done if blood cultures are repeatedly negative.

- **Blood picture:**

- ✓ Normocytic anemia.
- ✓ Polymorph nuclear leucocytosis in **ABE**.
- ✓ Leucopenia may occur in **SBE**.

- **ESR & C - Reactive protein:**

- ✓ Increased but not as markedly as in rheumatic fever.

- **Urine analysis:**

- ✓ Microscopic or macroscopic haematuria Proteinuria & casts.

- **Immunologic tests:**

- ✓ Increased gammaglobulins.
- ✓ False positive rheumatoid factor and serological tests for syphilis.

B. **Cardiac investigations:**

- **Echocardiography: (transoesophageal echo for small vegetation visualization).**

- ✓ Detects the underlying cardiac disease.
- ✓ Detects complication of endocarditis e.g. mitral & aortic incompetence.

- **Chest X-ray & ECG:**

- ✓ May help in diagnosis of the underlying cardiac disease.

Differential Diagnosis

1. Causes of fever in a cardiac patient especially rheumatic fever.
2. Causes of embolization.
3. Fever of unknown origin.

Duke's criteria for the diagnosis of infective endocarditis :-

Major criteria	1) Positive blood culture for infective endocarditis 2) Echo evidences of IE: vegetations - Abscess - New valve regurgitation.
Minor criteria	1) Predisposing factor: known cardiac lesion 2) Fever > 38 3) Echocardiogram : may be some findings but not as major. 4) Microbiological evidence: positive blood culture not as major criterion 5) Immunological : Roth spots , Osler nodes , GN , Rheumatoid factor. 6) Serological studies support an infection. 7) Vascular: emboli , mycotic aneurysm, conjunctival hemorrhage, Janeway L.

Clinical criteria for infective endocarditis requires:

- Two major criteria, or
- One major and three minor criteria, or
- Five minor criteria

Treatment**Prophylactic**

A. Correction of predisposing heart diseases: whenever possible

B. Prophylactic antibiotics:

Procedure done	Drugs	Before	After
Dental, oral, or U.R. surgery	Amoxicillin	3 gm 1 hour	1.5 gm 6 hours
	Erythromycin (Penicillin allergy)	1 gm 1 hour	0.5 gm 6 hours
GIT, or genitourinary	Ampicillin 2 gm	1 hour	6 hours
	Gentamycin 80mg IM-IV		
Heart s or catheter	Cefotaxime	2 gm, IV 2 hours	1gm / 6hours for 4doses

Therapeutic**1. Medical treatment:**

- Once infective endocarditis is suspected, treatment should be started immediately.
- Treatment should be continued for at least 6 weeks or longer in severe cases.

a. Antibiotic regimens include:

classic

For S.B.E.: (usually due to *Strept. Viridians* & *faecalis*):

Soluble Penicillin G 2-4 million units / 6h IV or Ampicillin 2 gm / 4h IV
Plus gentamycin: 80 gm/8 h IM.

- **For ABE:** (usually due to *Staph. aureus* & Gram – negative bacilli)
As in SBE with addition of Cloxacillin: 2 gm/4 h IV.

N.B.

For penicillin-allergic patients: Vancomycin is given.

- **Resistant cases:** Should be treated according to results of blood culture.
- **If fungal endocarditis is suspected:** (Amphotericin B given)

b. Symptomatic treatment:

- Rest: complete rest in bed until clinical manifestations improves.
- Diet: light nutrient diet
- Fever: e.g. antipyretics
- Treatment of complications: e.g. Heart failure Renal failure

N.B.

- *Anticoagulant therapy: has not been shown to prevent embolization in infective endocarditis and may increase the risk of intra-cerebral hemorrhage (Q; mycotic aneurysm).*

2. Surgical treatment:**Indications:**

- Valve replacement in : severe valvular damage.
- Replacement of infected prosthesis.
- Excision of infected ductus arteriosus or coarctation.

N.B.

- Any underlying infection should be treated e.g. **dental abscess**.

Differentiation between Rheumatic Fever & Infective Endocarditis:-

	<i>Rheumatic fever</i>	<i>Infective endocarditis</i>
Fleeting arthritis	+Ve	-
Pericarditis	+Ve	-
Chorea	+Ve	-
S.C. nodules	+Ve	-
Erythema marginatum	+Ve	-
Petechiae	-	+Ve
Clubbing	-	+Ve
Osier's nodules	-	+Ve
Splinter haemorrhage	-	+Ve
Absent pulse	-	+Ve
Splenomegaly	-	+Ve
Kidney manifestations	-	+Ve
Embolic manifestations	-	+Ve
Blood culture	-	+Ve
echocardiography	No vegetations	Vegetations

Symptomatology

Dyspnea

Definition:- Uncomfortable awareness of breathing or Shortness of breath

Pathogenesis of Cardiac Dyspnea:

A. Mechanical factors

CVS	BV:- Pulmonary congestion Alveolar:- Intra-alveolar oedema Bronchial:- Ms:- weakness of respiratory muscles Pressure manifestation	LVF, MS, MR, AS, AR APO Oedema of bronchial mucosa Low COP Pericardial eff. - Huge cardiomegaly
Lung	BV:- Bronchial:- Pressure manifestation	- P. embolism - P. hypertension - Bronchial spasm - Pleural effusion - c
Abd.	Infra-diaphragmatic	Ascites - enlarged tender liver (RVF)

B. Nervous factors:

Activation of Hering-Breuer reflex (physiological)

Stretch receptors stimulated (at the end of inspiration) -----reflex inhibition of inspiration ----- expiration ----- *shallow rapid respiration.*

Churchill-C one reflex : (not physiological)

P. congestion ----- BV receptors ----- respiratory centre ----- tachypnea.

C. Chemical factors:

- Hypoxia occurs due to low CO & pulmonary congestion.
- $> \text{CO}_2$ + Acidosis occurs due to tissue hypoxia.

Types of Cardiac Dyspnea:

A. Exertional dyspnea: NYHA

- * Grade I: dyspnea on more than ordinary effort (severe exertion).
- * Grade II: dyspnea on ordinary effort (moderate exertion).
- * Grade III: dyspnea on less than ordinary effort (mild exertion)
- * Grade IV : dyspnea at rest.

B. Orthopnea: Dyspnea on lying flat which relived by sitting.

- Pathogenesis:-**
- 1- Increased **venous return** aggravates pulmonary congestion.
 - 2- Elevation of the **diaphragm**.
 - 3- Interference with the action of **respiratory muscles**.

C. Paroxysmal Nocturnal Dyspnea (PND)

Clinical picture:

The patient arises **1-2 hours** after sleep with severe **inspiratory dyspnea**, cough $\pm$ frothy expectoration. _ wheez maybe present (bronchial oedema & bronchospasm).

The condition is relieved **spontaneously** or with treatment - but may be recurrent

Pathogenesis (1-3 as Orthopnea)

1. Increased venous return aggravates pulmonary congestion.
2. Elevation of the diaphragm.
3. Interference with the action of respiratory muscles.
4. Absorption of oedema fluid into the circulation (VR).
5. Decreased sympathetic activity during sleep (reduction of contractility).
6. Nightmares: may lead to tachycardia & elevation of BP (load).
7. Slipping down from high pillows.

} as Orthopnea

- Orthopnea is not specific to cardiac diseases
- PND is a specific symptom of left sided heart lesion especially LSHF

* We have to exclude B.A.

Differentiation between Cardiac & Bronchial Asthma

	Cardiogenic	Bronchial
Age	Any age	Usually young age
Past history	Cardiac symptoms	Chest symptoms
Duration	Usually short	Usually long
Time of attack	1-2 hours after sleep	Early in the morning
Dyspnea	Mainly inspiratory	Mainly expiratory
Sputum	Frothy & may be bloody	Thick pellets
Chest exam.	Basal crepitations	Wheezes
Heart exam	Gallop & murmurs	Normal
ECG	Abnormal	Normal
Effect of drugs		
Adrenaline	Contraindicated	Improves the condition
Morphine	Improves the condition	Contraindicated
Aminophyllin	Improves the condition	Improves the condition

OEDEMA

Definition:- Accumulation of excessive fluid in the interstitial spaces

Pathogenesis of oedema:**1. Increased capillary hydrostatic pressure:**

- Increased venous pressure: HF, Pericardial effusion, constrictive Pericarditis, Venous occlusion e.g. thrombosis.
- Salt & water retention.

2. Decreased plasma osmotic pressure : (Due to hypoproteinaemia)

- Malnutrition.
- Malabsorption.
- Liver failure.
- Nephritic syndrome.

3. Increased capillary permeability:

- Inflammation: e.g. Infection, Allergy, Chemicals or Trauma.
- Hypoxia

4. Lymphatic obstruction

Generalized Oedema:

- **Cardiac:** Right-sided heart failure, pericardial effusion & constrictive pericarditis.
- **Hepatic:** Liver failure.
- **Renal:** Nephrotic syndrome & Nephritic syndrome.
- **Nutritional:** Malnutrition, Malabsorption.
- **Severe allergy.**

Pathogenesis of Cardiac Oedema:**A. Increased capillary hydrostatic pressure:**

1. Increased venous pressure: due to stagnation of blood in the systemic veins.
2. Salt & water retention (see later)

B. Hypo-albuminaemia:

1. Diminished protein **intake** due to anorexia & vomiting.
2. Diminished protein **absorption** due to intestinal congestion
3. Diminished albumin **synthesis** in the liver
4. Increased protein **loss** in urine.

C. Increased capillary permeability: due to hypoxia.**D. Salt & water retention:**

1. Decrease Glomerular filtration rate & urine output : (Low cardiac output).
2. Secondary hyperaldosteronism:-
 - Secondary to low CO (reduction of renal blood flow ----- rennin)
 - Diminished destruction -----congested liver.
3. Increased ADH : due to :
 - Increased production secondary to low CO - Diminished destruction.
4. Decreased secretion of renal prostaglandins.

Features of Cardiac Oedema:-

1. **Dependent** : Oedema of lower limbs (Sacral oedema: bed-ridden patients).
2. **Bilateral & equal**: however, may be more in one side due to DVT, Postural (lye on one side).
3. **Lower limbs precedes ascites**: however, ascites may be the first " Ascites precox".
4. **Pitting & not tender**.
5. **Associated** with other features of cardiac disease.

Ascites precox:

- a. Pericardial effusion & constrictive pericarditis: due to **narrowing** of hepatic veins --- marked liver congestion----- **obstruction of lymphatics**.
- b. TR & TS: because regurgitation of blood causes marked liver congestion.

Causes of Local Oedema

1. Venous obstruction.
2. Lymphatic obstruction.
3. Inflammatory oedema.

Syncope

Definition:- Transient loss of consciousness due to acute cerebral ischaemia. If prolonged, convulsions may occur. Further prolongation causes coma, brain damage & death.

Pathophysiology:- Decreased cerebral perfusion is the final common pathway leading to syncope.

Clinical picture:- Patients may describe a syncopal episode in many ways, including blackout, dizzy spell, and seizure or unexplained falls, particularly in elderly persons.

Aetiology

1. Vasomotor syncope (the commonest)

A- Vasovagal syncope (neurogenic syncope): (known as simple fainting)

- Severe vagal stimulation --- severe bradycardia, hypotension, pallor & sweating.

- **Causes:** sudden severe fear, pain, and trauma (e.g. to testicles).

B- Carotid sinus syndrome:

Pressure on hypersensitive carotid sinus baroreceptors: e.g. during shaving.

2. Cardiac syncope (Any cause of low CO) especially:

- Aortic stenosis (or any other valvular obstruction).
- Acute heart failure (e.g. AMI).
- Arrhythmias (whether tachy- or brady-arrhythmia).
- Adams-stokes attacks.

- Usually exertional.
- Not convulsions.

3. Cerebral syncope (Reduced cerebral blood flow)

- Vertebrobasilar TIAs.

4. Hypoxic syncope (↓ O₂ content of the cerebral blood flow)

- Fallot's tetralogy & other cyanotic diseases or severe anemia.

5. Postural syncope (Orthostatic syncope)

During standing:- sympathetic ---- reflex VC of BVs of L.Ls ----- prevent pooling of blood in lower limbs. failure of this mechanism leads to postural hypotension

Causes:

Reduce Sympathetic	- Autonomic neuropathy, e.g. Diabetes. - Sympatholytic drugs, e.g. ganglion blockers & vasodilators. - Lumbar sympathectomy.
Reduced Volume	- Hyponatremia - Hypovolaemia (Haemorrhage or dehydration) - Huge varicose veins.
Weak LL Ms	- Prolonged recumbency - Elderly patient. - Weakness of the muscles of the lower limbs (muscle pump).

6. Situational syncope

- Cough syncope (*Tussive syncope*).
- Micturition syncope (*more common in old men especially at night*).
- Defecation syncope.

explained as:- Straining → decreased VR → decreased CO → syncope.

Cyanosis / palpitation / Jaundice and Fever in cardiac patient:- see clinical book

Haemoptysis in cardiac patients

Causes	Features
– Acute pulmonary edema. – Pulmonary infarction. – Pulmonary infection. – Pulmonary apoplexy. – Severe hypertension. – Ruptured aortic aneurysm into a bronchus. – Associated conditions causing haemoptysis	– Frothy blood-tinged sputum. – Frank haemoptysis. – Blood-streaked mucopurulent. – Profuse frank haemoptysis (rare).

It may occur due to rupture of bronchial varices occurring in chronic mitral stenosis.

Cardiovascular causes of chest pain

Origin	Causes	Cardiac neurosis:
Myocardium	IHDs: angina - MI.	Neurotic patients especially females. Pain:- Localized left inframammary. Stitching, of variable duration. No relation to exertion. -Not relieved by rest. Associations other neurosis. Normal cardiac examination.
Pericardium	Pericarditis - pericardial eff.	
Endocardium	Mitral valve prolapse.	
Aorta	Aortic aneurysm - dissection.	
Pulmonary	P. embolism.	
Huge cardiomegaly:- Rare		

Valvular heart diseases

IN ANY VALVE LESION

1- Aetiology:-

	Stenosis	Regurge
Usual	- Rheumatic fever. - Congenital	
Spec.	Calcific	- Infective endocarditis - Surgical
Relative	- Relative:- increased B flow	- Functional (Dilatation of the ring)

Acute valve Regurge :- Surgical - infective endocarditis + (IHDs in MR-TR)

2- Pathophysiology:- haemodynamic change → C/O- General-Precordial ex

3- Clinical picture:-

- C/O:-**
- Aorta = Chest pain
 - Regurge = palpitation (NB: AF = Irrg. palpitation)
 - MS = 1st P. congestion

Signs

A- General:-

- Pulse - ABP
- Peripheral sign = AR
- NVs = Right sided (PH++ - TR - RVF)
- Signs of
 - P. congestion=BBC
 - S. congestion
 - Low COP

B- Cardiac:

A. Precordial examination (inspection-palpation-percussion):

1. Chamber +++
2. Apex
3. Thrill

B. Auscultation: you must mention over which area

- 1- HS
- 2- Add. S
- 3- Murmur 5 (time-chr-site- propagation-how to increase)

4- Complications

1. HF
- 2- Infective endocarditis
- 3- Rheumatic activity

5- Investigation:-

X-ray	ECG	Echo (the most diagnostic)	Catheterization
- Chamber ++ - P. vasculature - Calcific Valve	- Chamber ++ - Arrhythmia - IHDs	Aetiology - lesion Function (EF) - Complications - Severity ?????	As Echo + therapeutic

6- Treatment :-

- Medical
- Interventional
- Surgical

Mitral stenosis (MS)

Aetiology:-

1. Rheumatic fever:- (the most common cause).
2. Congenital: a rare cause.
3. Relative stenosis (not an organic stenosis)
 - Increased blood flow through the mitral valve.
 - Carey-coomb's murmur in acute rheumatic valvulitis.
 - Austin flint murmur in severe aortic regurgitation.

Pathophysiology

- In mild cases:
The blood flow through the mitral valve remains normal. No symptoms occur.
- In severe cases: (valve area less than 2 cm^2): Decreased blood flow.
Blood stagnate in pulmonary veins (**pulmonary venous congestion**).
- Later on:
Vasoconstriction of pulmonary arterioles --- ↓ P. congestion + **Pulmonary hypertension**.
- Finally:
RV enlargement & then RVF will occur secondary to pulmonary HTN.

Therefore four stages will occur in patient with MS

Stages of Mitral stenosis

- **Stage one:** "Mild or asymptomatic mitral stenosis"
The only abnormality is anatomical narrowing of the mitral valve, but no symptoms.
- **Stage two:** "mitral stenosis with pulmonary congestion".
- **Stage three:** "mitral stenosis with pulmonary hypertension"
- **Stage four:** "mitral stenosis with right ventricular failure"

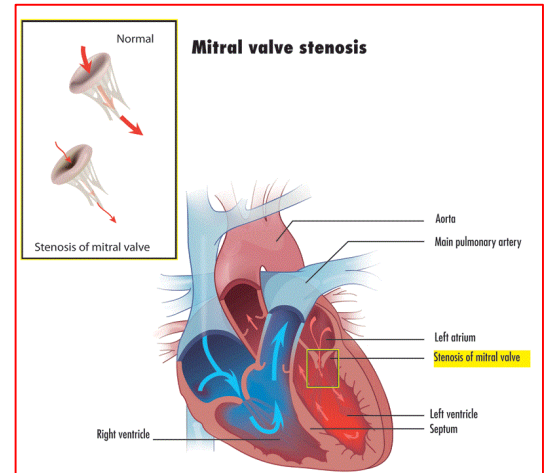
Clinical picture

There is a **latent period** of several years between the initial attack of rheumatic carditis & the development of manifestations of mitral stenosis.

Symptoms Does the patient with MS always symptomatize???

Stage one:	No symptoms.
Stage two:	Symptoms of pulmonary congestion (but pulmonary oedema is not common). Symptoms of low CO.
Stage three:	Symptoms of pulmonary congestions: improve. Symptoms of Low CO: increase.
Stage four:	Symptoms of systemic congestion

Dyspnea is the most common symptom



Signs**General:**

Stage one:	No signs.
Stage two:	signs of pulmonary congestion .
Stage three:	signs of low cardiac output .
Stage four:	signs of systemic congestion .

Cardiac:**A. Precordial examination:**

Stage one:	Apex: normal site & slapping character.
Stage two:	Diastolic thrill : ending in a palpable S1.
Stage three:	The previous findings.
Stage four:	+ Signs of: Pulmonary hypertension . Signs of: RV enlargement.

B. Auscultation (Summary)

Stage one:	Over Mitral area
Stage two:	<u>HS:-</u> * Accentuated S1 <u>Add. Sound:-</u> * Opening snap <u>Murmur:-</u> Mid-diastolic with presystolic accentuation - rumbling - increased on left lateral position
Stage three:	Over Pulmonary area <u>HS:-</u> Accentuated S2 <u>Add. Sound:-</u> Ejection click + S4 (Over T area) <u>Murmur:-</u> Systolic ejection murmur. - Soft early diastolic murmur: (Graham Steel murmur)
Stage four:	As in RV failure:- Over Tricuspid area S3 gallop + Functional TR pansystolic murmur

Stage one & stage two (over the mitral area)1. Accentuated first heart sound: due to:

- + Fibrosis of the mitral cusps.
- + Forcible closure of the mitral cusps: because:
 - ❖ They are displaced downwards due to high LA pressure.
 - ❖ The mitral valve is opened as wide as possible during the diastole.

N.B. *Diminished S1 in mitral stenosis denotes:*

- Double mitral lesion (with predominant regurge) - Calcified mitral valve.

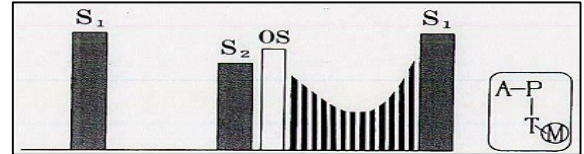
2. Mitral opening snap:

- It is **sharp & snapping** due to opening of the rigid cusps of mitral valve.
- It heard in the **early diastole**.
separated from S2 by the isometric relaxation phase - Just before the murmur.

Its Significance:- Sure MS - **non-calcific** valve - severity

3. Murmur of mitral stenosis:

- Timing:
 - * Mid-diastolic pre-systolic with pre-systolic accentuation.
 - * In AF, there is loss of pre-systolic accentuation (loss of atrial contraction).
- Character: rumbling.
- Site: at or slightly inside the apex.
- Propagation: not propagated.
- Position:
 - * Best heard with the cone of the stethoscope.
 - * In the left lateral position.



Silent mitral stenosis: (MS with no murmur) due to:

- 1- High LV pressure: LVF.
- 2- Low LA pressure (- Severe pulmonary HTN. - RVF).
- 3- In association with ASD.

Stage three

- The previous findings.
- Auscultatory findings of pulmonary hypertension:
 - a. Over the pulmonary area:
 - S2 is accentuated.
 - Systolic ejection click.
 - Systolic ejection murmur.
 - Soft early diastolic murmur: (Graham Steel murmur)
 - b. Over the tricuspid area:
 - S4 gallop

Stage four

- The previous findings.
- Auscultation over tricuspid area:
 - o Pan systolic murmur of functional tricuspid regurge.
 - o Proto-diastolic gallop (S3) due to RVF.

Complications

1. In the mitral valve:

- o Rheumatic activity.
- o Calcification.
- o Infective endocarditis.

2. In the left atrium:

- a) Arrhythmias, especially AF.
- b) LA enlargement, causing symptoms:
 - On esophagus: dysphagia.
 - On left bronchus: dyspnea & cough.
 - On left recurrent laryngeal nerve: hoarseness of voice.

c) Thrombo-embolic complications:

- Systemic embolization: e.g. Cerebral, peripheral, renal.
- Ball & valve embolus: leading to syncope & sudden death.

3. **In the right ventricle:**

- RVF.

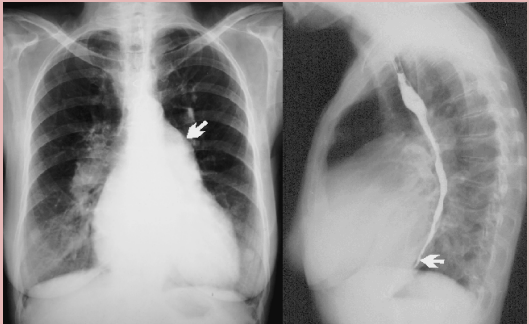
4. **In the left ventricle:**

- No LVF in isolated mitral stenosis.

5. **In the lung:**

- Hemoptysis.
- Pulmonary infection.
- Pulmonary embolism (secondary to DVT).
- Pulmonary oedema is not common in mitral stenosis.

6. **Complications of surgery.****Investigation:-**

X-Ray	Stage one: no abnormality. Stage two: Mitralization (LA++) - Obliteration of the waist - Double contour of the right border - Pulmonary congestion Stage three & four: - Right ventricular enlargement. - Pulmonary hypertension. - May be calcified mitral valve.	
ECG	Stage one: no abnormality. Stage two: LA +++ (P mitrale: broad & bifid). Stage three & four: RA +++ (P pulmonale: tall & peaked). + RV+++	
Echo	The most sensitive & specific non-invasive method diagnosing MS. - Detects the severity (Valve area < 1 cm²) - Chamber enlargement - function (EF)	
Catheter	AS ECHO	

Ba Swallow:- Enlarged left atrium displaces the esophagus posteriorly.

Tight MS is diagnosed:

- o **According to the stage:**
 - Stage two with dyspnea more than grade two, or
 - Stage three, or Stage four.
- o **According to the echocardiography:**
 - Valve area less than 1 cm².

Treatment

1. Medical:

- a) Prophylaxis against: rheumatic activity, infective endocarditis (uncommon).
- b) Symptomatic for: complications **e.g.** HF, AF, infection, embolization.

2. Surgical:

- a) Indications:
 - o Tight mitral stenosis (valve area is $< 1 \text{ cm}^2$).
 - o Marked symptoms not responding to adequate medical treatment.
 - o Embolization with no serious deterioration of the condition of the patient.
- b) Types of operations:
 - i. Mitral commissurotomy: closed or open (Old regimen of limited indications)
 - ii. Valve replacement: By a prosthesis (tissue or synthetic).
 - in - Calcific valve - Associated mitral regurge. - Failed commissurotomy.
- c) Complications:-
 - 1. Embolization.
 - 2. Arrhythmias.
 - 3. Mitral incompetence.
 - 4. Restenosis.
 - 5. Post-cardiotomy syndrome: Pleuro-pericarditis 10 – 15 days post-operative as an allergic process due to injury of the pericardium during surgery.
 - 6. Complications of artificial valves:
 - o Infective endocarditis.
 - o Thrombo-embolism.
 - o Mechanical dysfunction.
 - o Hemolytic anemia.

3. Interventional:- Balloon dilatation:

- May be performed in some patients indicated for valvotomy.

Mitral regurgitation (MR)

Aetiology

A. Organic:

1. Rheumatic fever: the most common cause.
2. Congenital.
3. Prolapse of the mitral valve (MVP)
4. Papillary muscle dysfunction **e.g.** CAD.
5. Infective endocarditis.
6. Iatrogenic: following mitral valvotomy.

Acute MR:-

- 1- post-valvotomy
- 2- Severe inf endocarditis (perforation)
- 3- acute papillary Ms. rupture (MI)

C/p:- presented with APO

- #### B. Relative:-
- Dilatation of the mitral ring secondary to LV dilatation.

Pathophysiology

1. During systole: Part of blood regurgitates from LV to LA leading to:
 - Low CO.
 - LA dilatation (blood from 2 sources)
2. During diastole:-
A large volume of blood → LV → LV enlargement which may end in LVF.

Clinical picture

Symptoms

1. No symptoms: in early cases.
2. Symptoms of low cardiac output: in late cases.
3. Symptoms of pulmonary congestion: in late cases.
4. **PALPITATION.**

Palpitation is the most common

Signs

General:

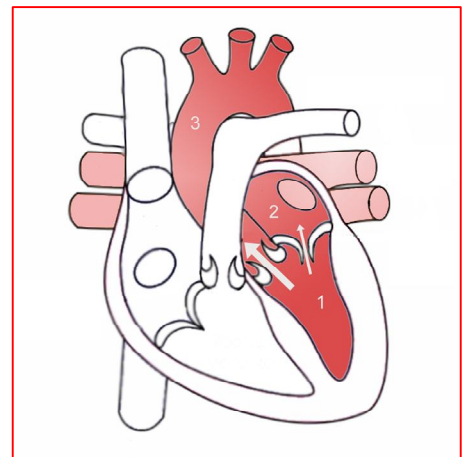
- * No signs: in early cases.
- * Signs of low cardiac output: in late cases.
- * Signs of pulmonary congestion: in late cases.

Cardiac:

A. Precordial examination:

- * Signs of LV enlargement: with hyperdynamic apex.
- * Systolic thrill: over the apex.

B. Auscultation:



HS	Weak S1 (muffled) due to failure of proper mitral closures
Add S	S3 (excessive flow of blood from LA to LV)
Murmur	<u>Timing</u>: pansystolic starting with S1. <u>Character</u>: soft or harsh. <u>Site</u>: over the apex. <u>Propagation</u>: - To the axilla - - To the base of the heart & medially in posterior leaflet disease. <u>Position</u>: heard best in the left lateral position

Complications

Same complications of MS, but:

1. Infective endocarditis: is common.
2. Left ventricular failure: occurs.

Investigations

X-Ray	* No abnormality: in early cases. * Enlarged LA & LV: in late cases. * Pulmonary congestion: in late cases. * Calcified mitral valve especially: in double mitral lesion.
ECG	* Enlarged LA (P mitrale:- broad & bifid). * Enlarged LV.
Echo	* Detects the severity of mitral regurgitation. * Detects chamber enlargement. * Detects the cause: e.g. MVP.
Catheter	AS ECHO

Treatment

1. **Medical:** Same as that of mitral stenosis.
2. **Surgical:** Valve replacement.

Mitral valve prolapse (Barlow's syndrome – Floppy mitral valve)

- It is common in young females.
- Mild prolapse is regarded as a normal variant.

Aetiology

1. **Unknown**, but there is familial incidence.
2. Isolated abnormality, **but** may be associated with: **Marfan's syndrome or ASD**.

Pathophysiology

During systole, a mitral valve leaflet (commonly the posterior) prolapses into the L.A.

This may result in papillary muscle strain & some mitral regurge.

Usually the case is not hemodynamically significant.

Clinical pictures

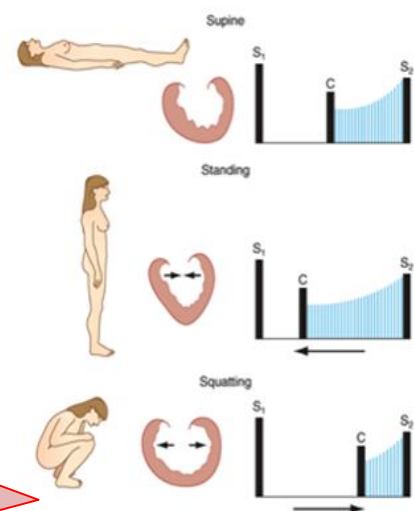
Symptoms

- * Atypical chest pain: is the most common symptom.
- * Palpitation.

Signs

- * Mid systolic click.
- * Late systolic murmur.

In contrast to most other heart murmurs, the murmur of mitral valve prolapse is accentuated by standing and valsalva maneuver



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: Harrison's Principles of Internal Medicine, 18th Edition: www.accessmedicine.com
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Complications

- * Progressive mitral regurgitation.
- * ***Infective endocarditis.***
- * Systemic embolization.
- * Arrhythmias.
- * Sudden death.

Investigations:- *Echocardiography* is diagnostic.

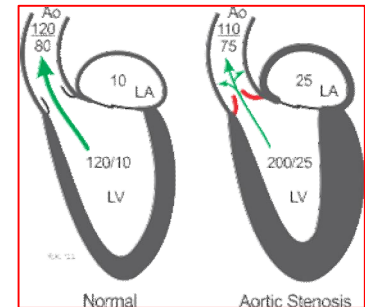
Treatment

- * Reassurance.
- * Prophylaxis against: ***infective endocarditis.***
- * Anti-arrhythmic therapy.
- * ***Propranolol:*** is effective for chest pain.
- * Surgery: mitral valve replacement in cases with severe MR

Aortic stenosis (AS)

Aetiology

1. Rheumatic fever.
2. Calcific.
3. Congenital:
it may be: valvular, subvalvular or supra-valvular.
4. Hypertrophic cardiomyopathy (Idiopathic Hypertrophic Subaortic Stenosis; IHSS):
It produces a subvalvular obstruction in the systole due to: contraction of the hypertrophied interventricular septum.
5. Relative stenosis (not an organic stenosis):
 - a. Dilatation of the aorta:
Hypertension, atherosclerosis, aortic aneurysm.
 - b. Increased blood flow across the aortic valve:
Hyperdynamic circulation, AR.



Pathophysiology

- During systole, there is obstruction of blood flow from LV to aorta leading to:
 1. Low cardiac output.
 2. Pressure overload on LV leading to LVH & LVF.
- Normally, the aortic valve area is 3 – 4 cm². In severe AS, it is less than 0.8 cm².

Clinical picture

Symptoms

1. No symptoms: in mild cases.
2. Symptoms of low CO: in severe cases.
3. Symptoms of pulmonary congestion: due to LVF.
4. SYNCOPE: especially **exertional** due to low fixed CO.
5. ANGINA: due to
 - Reduced coronary blood flow: due to low CO & shortened diastole.
 - Left ventricular hypertrophy: increases the myocardial O₂ demands.
 - Associated coronary atherosclerosis: especially in calcific AS.

Signs

General:

1. Pulse:
 - * Pulsus parvus et tardus (plateau pulse): *rises slowly, of small volume, returns slowly.*
 - * Pulsus bisferiens: *bifid pulse occurring in double aortic lesion.*
2. BP: low SBP in severe cases.
3. Systolic thrill: over the carotid arteries.

Cardiac:**C. Precordial examination:**

4. Signs of LVH: with a heaving apex.
5. Systolic thrill: over second right space.

D. Auscultation:Over the aortic area:

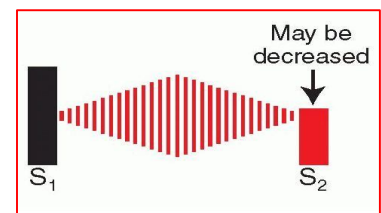
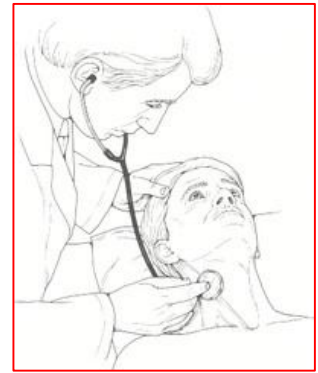
1. Second heart sound: weak.
2. Systolic ejection click: due to opening of the rigid cusps.
3. Systolic ejection murmur:
 - Midsystolic, harsh.
 - Maximum over second right space → apex & carotid arteries.

Over the pulmonary area:

- Reversed splitting of the second heart sound.

Over the mitral areas:

1. S₄.
2. Propagated murmur of AS.

**complications**

2. LVF.
3. Infective endocarditis.
4. Sudden death: usually due to VF.
5. Heart block: in calcific AS due to extension of calcification to AV bundle.
6. Rheumatic activity: in rheumatic AS.

Investigations

X-Ray	No abnormality: in mild cases. LV: LVH. Lungs: pulmonary congestion when LVF occurs. Aorta: Small aortic knuckle or post-stenotic dilatation. Aortic valve calcification may be seen.	
ECG	* Enlarged LV.	
Echo	* Detects the severity of stenosis by: - Measurement of valve area. - Measurement of pressure gradient across the valve. * Detects the type of stenosis. * Detects LVH	
Catheter	AS ECHO	Tight AS:- 1- Pressure gradient > 50 mmHg 2- Valve area > 0.8 cm ²

Treatment

1. Medical:

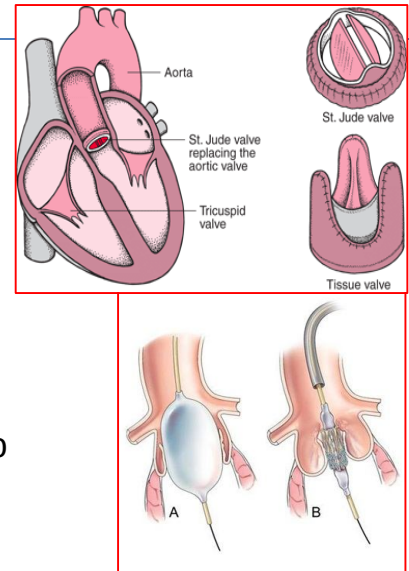
- Same as that of mitral stenosis.
- Anginal attacks: may be relieved by SL nitrates.

2. Surgical: (Aortic valve replacement) indications:

- Presence of severe symptoms.
- Pressure gradient more than 50 mmHg.
- Valve area less than 0.8 cm^2 .

3. Balloon dilatation: indications:

- Children:** with congenital AS as an alternative to surgery.
- Elderly:** with severe calcific AS who are too ill to undergo surgery.



	Congenital	Rheumatic	Calcific
Age	Young	Young & middle	Old
History of RF	Absent	Present	Absent
Type	Valvular or sub/supravalvular	Valvular	Valvular
Associations	Congenital anomalies	Other valvular lesions	Atherosclerosis

Aortic regurgitation (AR)

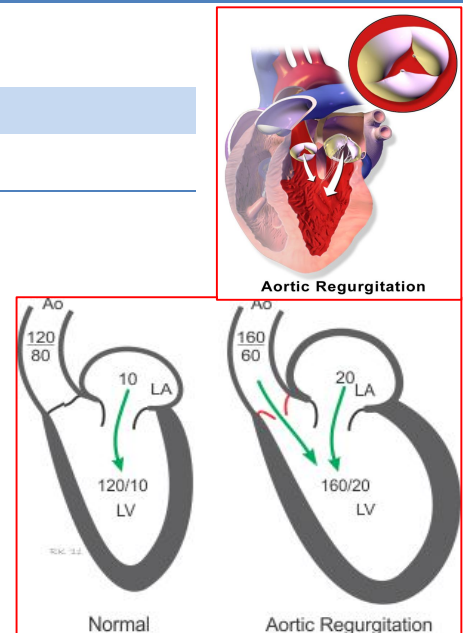
Aetiology

- Rheumatic fever: **the most common cause**.
- Infective endocarditis.
- Congenital.
- Dilatation of the ascending aorta as in :
 - Syphilitic aortitis (Dilated aortic ring).
 - Severe hypertension.
 - Ankylosing spondylitis.
 - Aortic aneurysm: with dissection.
 - Marfan syndrome.

Pathophysiology

Regurgitation of blood from the aorta to the LV in diastole:

- Increased LV stroke volume, this results in:
 - Increased SBP.**
 - Peripheral VD which (together with regurgitation) will **decrease the DBP.**
 - $\uparrow \text{SBP} \ \& \ \downarrow \text{DBP} \rightarrow$ wide pulse pressure causing peripheral signs of AR.
- Volume overload on the LV leading to LV dilatation & later on LVF.



Clinical picture

Symptoms

1. Generalized **body throbbing**: due to increased arterial pulsation.
2. **Palpitation**: due to forcible LV contraction.
3. Symptoms of pulmonary congestion: when LVF occurs.
4. Angina pectoris: "two types of angina occur in aortic regurge"
 - **Classic angina of effort** : Decreased DBP: reduces coronary filling.
 - **Angina of Lewis**:
Nocturnal & associated with autonomic disturbance **e.g.** sweating & tachycardia.

Signs

General: "Peripheral signs of aortic regurge"

1. De-Musset sign: nodding of the head.
2. Corrigan's sign: prominent carotid pulsations.
3. Systolic thrill: over the carotid arteries.
4. Water hammer pulse: rises rapidly, of big volume, collapses rapidly.
5. Capillary pulsations: detected in nail bed, lips or ear lobule.
6. Pistol shots: loud booming sounds heard with each pulse beat over the arteries (especially femoral) due to sudden distension of collapsed arteries.
7. Duroziez's sign:
 - Systolic & diastolic murmurs over the femoral artery after be compressed with the stethoscope bell.
 - The systolic murmur is due to the rapid flow of blood to periphery
 - The diastolic murmur is due to rapid regurge of blood to the heart.
8. Blood pressure:
 - a. Wide pulse pressure:
 - Exaggerated difference between systolic & diastolic blood pressure.
 - b. Hill's sign:
 - Exaggerated difference between SBP in LLs & ULs: more than 50 mmHg.
 - Normally, SBP in LLs is higher than in ULs: by about 10 – 20 mmHg.

Cardiac

- A. Precordial examination:
 - Signs of LV enlargement: with hyperdynamic apex.
 - No thrill over the aortic area: in isolated AR.
- B. Auscultation:
 - Over the aortic area:
 - a. Normal second heart sound.
 - b. Murmur of AR:
 - o Timing: early diastolic.
 - o Character: soft blowing, decrescendo.
 - o Site: maximum over the third space.



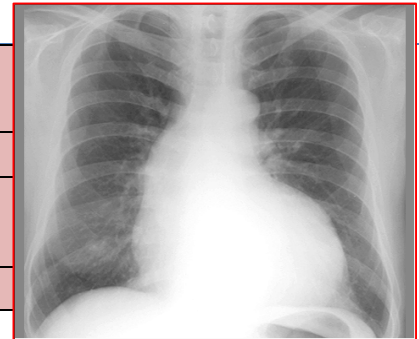
- o Propagation: to the apex.
- o Position: **best heard with the diaphragm of the stethoscope, the patient is:**
 - Sitting up. - Leaning forward. - Holding breath in forced expiration.
- c. Soft ejection systolic murmur:
 - o Due to ↑ blood flow across the aortic valve (relative AS).
- Over the mitral area:
 - a. S3.
 - b. *Propagated murmur: of AR.*
 - c. Pan systolic murmur: of functional MR.
 - d. **Austin Flint murmur:** (mid-diastolic) due to:
 - Elevation of anterior leaflet of the mitral valve by the regurgitant blood from the aorta causes relative **MS**.

Complications

1. Rheumatic activity.
2. Infective endocarditis.
3. LVF.

Investigations

X- Ray	"Aortic configuration (Boot-shaped heart)" - LV enlargement & dilated aorta.
ECG	* LV++
Echo	• Detects the severity of the lesion. • Detects chamber enlargement.
Catheter	AS ECHO



Treatment

1. Medical:

- Same as that of mitral stenosis.
- Syphilis: anti-syphilitic treatment.

2. Surgical: (aortic valve replacement) indications:

- a. Presence of severe symptoms.
- b. Progressive cardiomegaly.
- c. Declining LV functions.

Tricuspid stenosis (TS)

Aetiology

- Rheumatic fever: the most common cause, & *is almost always associated with M.*
- Carcinoid syndrome: a rare cause.

Pathophysiology

During diastole, there is obstruction of blood flow from RA to RV leading to:

- Systemic venous congestion.
- Low CO.

Clinical picture

Symptoms

1. Systemic venous congestion.
2. Low CO.

Signs

General:

1. Systemic congestion including:
 - Congested pulsating neck veins with giant A wave.
 - Enlarged tender pulsating liver.
 - Ascites **before** oedema of lower limbs (*ascites precox*).
2. Low CO.

Cardiac:

- A. Precordial examination:
 - RA enlargement (dullness to the right border of the sternum).
 - RV enlargement (due to frequently associated MS).
- B. Auscultation: *Over the tricuspid area:*
 - Accentuated first heart sound.
 - Opening snap.
 - Mid-diastolic murmur that increases by inspiration.

Investigations

X- Ray	- RA & RV enlargement
ECG	- RA & RV enlargement
Echo	• Detects the severity of the lesion. • Detects chamber enlargement.
Catheter	AS ECHO

Treatment

1. Medical: Treatment of right sided heart failure.

2. Surgical: Valve replacement.

Tricuspid regurgitation (TR)

Aetiology

1. Functional: **the most common cause** due to dilatation of the tricuspid ring secondary to RV dilatation as **M.S.**
2. Organic: **rare:**
 - Rheumatic fever.
 - Infective endocarditis.
 - Congenital.
 - Carcinoid syndrome.

Pathophysiology

During systole, blood regurgitates from RV to RA causing **(V. overload)**

- Low CO.

+ - RA enlargement. - RV enlargement. - RVF (S. congestion).

Clinical picture

Symptoms

1. Systemic congestion.
2. Low CO.

Signs

General:

1. Systemic congestion including:
 - *Congested pulsating neck veins: with systolic expansion.*
 - *Enlarged tender pulsating liver.*
 - *Ascites **before** oedema of LLs (Ascites precox).*
 - *Mild jaundice & peripheral cyanosis (cyano-icteric face).*
2. Low CO.

Cardiac:

- A. Precordial examination:
 - RA & RV enlargement.
 - Rarely: systolic thrill over tricuspid area.
- B. Auscultation: "over tricuspid area"
 - a. Weak muffled S1.
 - b. S3.
 - c. Pan systolic murmur.
 - Timing: pan systolic.
 - Character: soft or harsh.
 - Site: tricuspid area.
 - Propagation: to the apex, but not to the axilla.

- Caravalllo's sign: murmur increases with inspiration being of right side origin.

Investigations

X- Ray	- RA & RV enlargement
ECG	- RA & RV enlargement
Echo	• Detects the severity of the lesion. • Detects chamber enlargement.
Catheter	AS ECHO

Treatment**1. Medical:**

- Treatment of right sided heart failure.

2. Surgical:

- Valve replacement.

Pulmonary stenosis (PS)**A. Organic:**

- Congenital: the most common cause.
- Carcinoid syndrome: rare.

B. Functional:

- Pulmonary hypertension.

Pulmonary regurgitation (PR)**A. Functional:**

- Mostly due to **PH** which causes dilatation of the pulmonary ring.

B. Organic:

- Congenital.
- Carcinoid syndrome.

The heart with multivalvular lesions what to do??

First

Valuable information may be observed in JVP and in arterial pulse:

- Raised JVP with prominent systolic wave = tricuspid regurgitation.
- Corrigan's sign – waterhammer = aortic regurgitation.
- Pulsus bisferiens (double strokes): aortic stenosis + aortic regurgitation.

Second

Pick the very significant abnormal signs suggestive of one lesion and then confirm by auscultation *i.e. one lesion by one.*

1. **Hyperdynamic apex:** either aortic or mitral regurgitation:
 - **Aortic regurgitation:** after noting Corrigan's signs (vigorous pulsation of the carotids) – waterhammer pulse: big pulse pressure with very low diastolic < 60 mm.
Then confirm by listening carefully to soft blowing early diastolic murmur at the lower right parasternal area.
 - **Mitral regurgitation:** check by feeling a **systolic thrill** at the apex (patient lying on his left side after exercise)
Confirm by listening to pansystolic murmur maximal over the mitral.
A mid diastolic murmur may also be heard (mitral stenosis).
2. **Signs of pulmonary hypertension:**
 - Left parasternal heave. (RV +++).
 - Pulsations and dullness in second left space.
 - Diastolic shock. (accentuated P2).
 - Prominent "a" wave.

Practically mitral stenosis is highly suspected.

- Confirm then by listening carefully to a mid diastolic murmur commonly with a presystolic accentuation.
 - Otherwise suspect other causes of pulmonary hypertension.
3. **Heaving apex:** aortic stenosis or severe hypertension.
Aortic stenosis check with feeling a systolic thrill: patient sitting and leaning forwards, holding his breath in expiration.
Confirm by listening to a harsh ejection murmur over aortic area propagated to the root of neck.
Then feel the arterial pulse and revise its character: slow and prolonged.
 4. **Tricuspid regurgitation:** most easily suspected by noting:
 - The distended neck veins with marked systolic expansion (v wave).
 - Expansile liver (bimanual palpation).
 - Confirm by listening over tricuspid area for a pansystolic murmur which is increased by inspiration.

Valve lesion	Most important cause	Most important symptom	Most important sign	Chest X-ray	ECG
MS	Rheumatic	Pulmonary congestion esp. dyspnea	• Accentuated S1. • Mid-diastolic rumbling M over apex. • Pulmonary HTN	• Mitralization. • Pulmonary congestion.	• P-mitrale. • P-pulmonale. • AF.
MR	Rheumatic	Palpitation	• Pan systolic M & thrill over the apex propagated to axilla	• LAE. • LVE.	• LAE. • LVE.
AS	Rheumatic Calcified Congenital	Low CO esp. Angina & syncope	• Heaving apex. • Harsh ejection systolic M & thrill "over the second right space → carotid"	• LVE. • Small aortic knuckle or • Post-stenotic dilatation.	• LVE.
AR	Rheumatic Syphilitic	Throbbing palpitation	• Peripheral signs. • Hyper dynamic apex. • Early diastolic soft blowing M "over the third left space"	• Boot shaped heart.	• LVE.
TS	Rheumatic	Systemic congestion	• Giant a-wave. • Mid-diastolic rumbling M over tricuspid "↑ with inspiration"	• RAE.	• RAE.
TR	Functional	Systemic congestion	• Systolic expansion in neck vein. • Pansystolic M over tricuspid "↑ with inspiration"	• RAE. • RVE.	• RAE. • RVE.

SYSTEMIC HYPERTENSION

- It is defined as **persistent** elevation of arterial blood pressure.
- Diastolic BP is higher than **90 mm Hg** and or the systolic BP is higher than 140 mm Hg.
- The measurement of BP should be done at least **twice** on at least two subsequent visits.
- Under complete mental & physical **rest**.

Classifications of Hypertension

A. Systolic & diastolic hypertension

B. According to aetiology:

1. Essential hypertension.
2. Secondary hypertension.
3. Paroxysmal hypertension.
4. Hypertension in pregnancy.

C. According to clinical presentation

1. Benign, accelerated & malignant hypertension.
2. Uncomplicated & complicated hypertension.
3. Mild, moderate, severe & very severe hypertension.

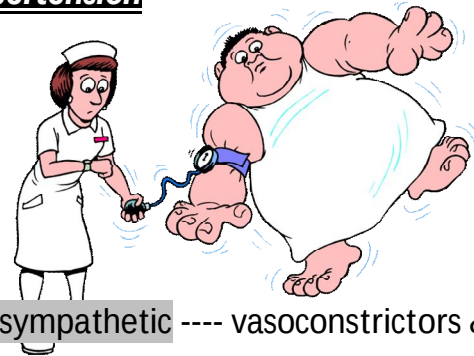
Isolated systolic hypertension:

- Aortic atherosclerosis
- Aortic incompetence
- Hyperdynamic circulation
- Marked bradycardia

Systolic & diastolic hypertension:

Essential (Primary) hypertension

- 95% of all cases.
- Middle & old ages (> 40) with family history
- Predisposing factors:
 - Heredity.
 - Obesity.
 - High salt intake.
 - Stress.



Theories of the aetiology include

Sympathetic over activity: excessive stimulation of sympathetic ---- vasoconstrictors & increases peripheral vascular resistance.

Renin secretion: Increased rennin of unknown cause (loss of negative feedback)----- converts angiotensinogen -- angiotensin I----- vasoconstriction & secretion of aldosterone ----- salt & water retention.

Hormonal factors: increased secretion of mineralocorticoid (salt & water retention)

Decreased vasodilator substances: As, prostaglandins, nitric oxide

Multifactorial theory: Stress --- sympathetic ---- vasoconstrictors ----- increases peripheral vascular resistance ----- renal ischaemia ----- rennin ---- aldosteron & VC

Others: eg - Hyperinsulinemia - Decreased atrial natriuretic peptide (ANP) - Baroreceptors resetting

Secondary Hypertension (curable hypertension)**Renal causes:**

Parenchymatous	Vascular (renal artery stenosis)
Reduced GFR --- Volume dependant HTN	renal ischaemia ----- renin-angiotensin system
– Acute & chronic glomerulonephritis. – Chronic pyelonephritis. – Hydronephrosis. – Polycystic kidney. – Diabetic nephropathy. – Collagen diseases e.g. SLE. PAN. – Gout nephropathy. – Nephrocalcinosis. – Amyloidosis.	– Atherosclerosis of renal artery. – Fibromuscular dysplasia. – Vasculitis e.g. polyarteritis nodosa (PAN). – Renal artery thrombosis & embolism.

Endocrinal causes:

- Cushing's syndrome & Conn's syndrome
- Pheochromocytoma
- Toxaemia of pregnancy & gestational hypertension
- Myxoedema
- Hyperparathyroidism
- Acromegaly

Neurological causes

- Increased intracranial tension.
- Lesions of the medulla e.g. bulbar poliomyelitis.
- Lesions of the hypothalamus.
- Polyneuritis: acute porphyria, chronic lead poisoning.
- Familial dysautonomia.

Drugs

- Contraceptive pills.
- Corticosteroids & ACTH.
- Cyclosporine.
- Carbenoxolone.
- Liquorice.

Miscellaneous causes

- Coarctation of the aorta.
- Polyarteritis nodosa.
- Polycythaemia vera.
- Hypercalcaemia.

Complications of Hypertension

1. Bleeding:

- Epistaxis (common), haematuria, retinal haemorrhage & cerebral haemorrhage.
- Haematemesis & haemoptysis (uncommon).

2. Cardiovascular:

Heart	Vascular
– LV hypertrophy (Pressure overload). – LVF. – Diastolic dysfunction of the left ventricle.	– Atherosclerosis of coronary. – Aortic dilatation with functional AR. – Aortic dissection.
– Bernhiem effect: concentric hypertrophy of LV may cause bulging of the septum in RV cavity leading to mild impairment of RV filling	

3. Vascular:-

- Atherosclerosis (Coronary, Cerebral or Peripheral vascular).

4. Cerebral:

- Cerebral atherosclerosis & thrombosis.
- Cerebral & subarachnoid haemorrhage.

	Hypertensive encephalopathy	Lacunar infarctions
Cause:	Sudden severe rise of BP	Prolonged rising of BP
Mechanism	Failure of autoregulation (VC)	Multiple small infarcts (1-5 mm)
Changes	Cerebral oedema ± petechial hemorrhages	(small cavities (lacunae))
Clinical	<u>ICT without signs of lateralization</u> Severe headache, vomiting & blurring of vision Drowsiness & convulsions Lastly coma may occur	May asymptomatic or mild <u>Focal neurological deficit</u> e.g. pure motor or sensory deficit, ataxia or dysarthria (according to the site)
lateralization	-Ve	+Ve

5. Renal:

- Renal failure: due to nephrosclerosis in **malignant hypertension**.
- Proteinuria.
- Haematuria.

6. Retinopathy: (4 Grades)

- **Grade I:** Mild **narrowing** of retinal arteries
- **Grade II:** Grade I + Marked sclerosis (**wiring**) & **kinking** of veins at A-V crossings.
- **Grade III:** Grade II + Flame-shaped **haemorrhage** and fluffy-cotton **exudates**.
- **Grade IV:** Grade III + **papilloedema**.

Clinical Picture of Hypertension**Benign hypertension:****Symptoms:**

- May be asymptomatic.
- Headache, tinnitus, dizziness, epistaxis & palpitation.
- Symptoms of complications.
- Symptoms of the cause in secondary cases.

Signs:

- **General:**
 - ✓ *Fundus examination:* for detection of changes hypertensive retinopathy.
 - ✓ *BP:* persistent elevation above 140/mm Hg.
- **Precordial examination:-**
 - ✓ Chamber: Left ventricular hypertrophy.
 - ✓ Apex: heaving.
 - ✓ Thrill: systolic.
- **Auscultations**
 - ✓ HS: Accentuated S2.
 - ✓ Additional S: Ejection systolic click.
 - ✓ Murmurs:
 - ❖ Ejection systolic murmur due to relative aortic stenosis.
 - ❖ Early diastolic murmur due to functional aortic incompetence.
- Signs of complications
- Signs of the cause in secondary cases.

Non-Benign hypertension:

Malignant hypertension:- Severe hypertension (diastolic BP > 130 mm Hg) with papilloedema.

Of abrupt onset, fulminated courses with severe generalized vascular damage & rapid severe complications (fatal) - RF ---- HF

Accelerated hypertension (pre malignant) as Malignant HTN without papilloedema
Retinal hrge or exudate (III) - If untreated, it usually progresses to the malignant HTN

Labile hypertension:- fluctuating HTN

Hypertensive crisis Marked increase of BP with Diastolic of >120-130 mm Hg.

It is classified into:

- **Hypertensive emergencies:** with ongoing target organ damage (TOD).
- **Hypertensive urgencies:** without ongoing target organ damage.

Investigations

1. For detection of associated risk factors of atherosclerosis:

- Blood glucose level.
- Lipid profile: cholesterol (LDL & HDL) and triglycerides.

2. For diagnosis of the cause

a. For renal parenchymal diseases

- Urine analysis.
- Renal function tests e.g. blood urea & serum creatinine.
- Renal imaging e.g. abdominal ultrasonography.

b. For renal vascular disease (see later)

c. For endocrinal diseases: (See Endocrinology)

3. For detection of complications of hypertension (Target organ damage) TOD

a. Cardiac Investigations:

- Chest X-ray: LV hypertrophy.
- ECG: LV and LA enlargement. May be evidence of IHDs.
- Echocardiography:- LV hypertrophy. Impaired diastolic function.

b. Neurological investigations: CT & MRI.

c. Renal investigations

Terms

Major risk factors

- Smoking.
- Dyslipidaemia.
- Diabetes mellitus (?????)
- Age > 60 years.
- Sex: men-postmen. women
- Family history of CV disease: women < 65 or men < 55 yrs.

CCD: clinical CVS disease

- LV hypertrophy.
- Angina or prior MI
- Prior coronary revascularization.
- HF.

TOD: Target organ damage

- Stroke or transient ischaemia.
- Nephropathy.
- Peripheral V disease.
- Retinopathy.

- * **Risk Group A:** No Risk Factors or TOD/CCD
- * **Risk Group B:** At least 1 risk Factor (Not DM).
- * **Risk Group C:** CTOD/CCD - DM ($\pm$ other risk Factors).

Classification of blood pressure measurements:

Category	Systolic BP	Diastolic BP
High normal	130 – 139	85 - 89
Hypertension		
Mild (Stage I)	140 – 159	90 - 99
Moderate (Stage II)	160 – 179	100 - 109
Severe (Stage III)	> = 180	> = 110

Strategy of treatment

Lifestyle modification:- FOR

- 1- high normal $\pm$ one risk factor
- 2- grade I HTN $\pm$ one risk factor
- 3- with all pharmacological ttt

Pharmacological TTT:- FOR

- 1- Failed lifestyle
- 2- Risk group C (CTOD/CCD or 2 risk factors)
- 3- Moderate or high BP (II-III)
- 4- Any DM

Treatment of Hypertension

A. Non-pharmacological Lines (Lifestyle modification)

- **Stop smoking**
- **Lose weight:** (aimed at BMI of 20 - 25 kg / m²).
- **Diet:** - Avoid foods high in **cholesterol** or animal fat - Reduce **salt** intake.
- **Regular exercise:** - Regular aerobic physical exercise (brisk walking not weightlifting).
- For 30 minutes daily or at least three times / week.

B. Pharmacological Lines:

Antihypertensive drugs

A- Diuretics

- 1- Loop:- Furosemide:- 40 – 160 mg / Oral / IV
- 2- Thiazides:- Hydrochlorothiazide - Indapamide
- 3- Potassium-Sparing:- Spironolactone.

Side effects :-

Hypo: K-Na-Ca-alkalosis-H₂O

Hyper: glycemia, lipidemia, uricemia
- ototoxic - nephrotoxic

B- Vasodilators

1- Direct Vasodilators

- **Hydralazine**:- 5 – 50 mg tds.
or 10 – 20 mg / 30 min IV (**h. encephalopathy**).
- **Nitroprusside**:- 0.5 – 2 ug / kg / min IV (**h. encephalopathy**)
- Diazoxide. - Nitrates

Side effects :-

- Tachycardia - Lupus like
- Precipitation of angina

2- Calcium Channel Blockers

- Verapamil: 80-120 mg/8 hrs orally
- Amlodipine: 5-10 mg / d orally.

Side effects :-

- Nausea and vomiting
- Photolytic

3- Angiotensin Converting Enzyme (ACE) Inhibitors

- Captopril 12.5 - 50 mg tsd
- Enalapril. - Ramipril (recent)

Side effects :-

- Leucopenia - Pancytopenia.
- Proteinuria - nephritis
- Fever and cough
Cl:- with creatinin > 2.8 mg%

4- Angiotensin-II Receptor blockers (ARBs).

- Losartan (**no cough but Hypersensitivity, hyperkalemia**)

C- Sympatholytics

a. Centrally acting drugs :-

- Clonidine (Catapres). - Methyldopa (Aldomet).

b. Ganglion blockers - Trimethopran.

c. Neurotransmitter:

- Inhibition of synthesis: alpha-methyl dopa.
- Release: Guanethidine.
- Depletion of stores: **Reserpine**.

Side effects :-

- Postural hypotension
- rebound hypertension
- dry mouth
- impotence

Side effects :- as above +

- suicidal tendency
- parkinsonism

d. Receptors antagonist

1. Alpha blocker:- Prazosin
2. Beta blocker
 - non selective:- propranolol
 - selective :- **atenolol** 50-100 mg/day orally
3. Alpha & Beta blocker:- Labetalol.

Side effects :- as above +

- Bronchospasm
- peripheral ischaemia
- depression
- Masking manifestations of hypoglycemia in DM

Tranquilizers Diazepam**Treatment of the cause in 2ry hypertension**

- Acute glomerulonephritis.
- Polycythaemia vera.
- Endocrinal causes e.g. Cushing's.
- Renal artery stenosis.

Treatment of complications

- Heart failure.
- Cerebrovascular stroke.

Treatment of hypertensive emergencies

- Hypertensive encephalopathy.
- Subarachnoid & cerebral haemorrhage.
- Acute left ventricular failure & pulmonary oedema.
- Myocardial infarction & ischaemia.
- Dissecting aortic aneurysm.
- Renal failure.

1. Treatment of Benign Hypertension

stepped care management :- The principle of treatment is to control BP using the fewest drugs in the lowest doses.

Diagram of stepped care management (not used)

Start with:	Single drug	Two drugs	Three drugs	Four drugs
	Thiazide or B-blocker or ACE inhibitor.			
	Uncontrolled D BP.	Add a 2nd drug of the previous		
		Uncontrolled BP.	Add the 3rd or Ca blocker or prazosin	
			Uncontrolled BP.	Add Frusemide or Guanithidine or Minoxidil

Individualized regimen of hypertension therapy

	Avoid	Allowed
Heart failure	• B-blockers. • Ca channel-blockers. • Guanithidine.	• Diuretics. • Vasodilators.
Ischaemic heart disease	• Hydralazine.	• B-blockers. • Ca channel-blockers.
Diabetes mellitus	• B-blockers. • Thiazides. • Frusemide.	• ACE inhibitors. • Ca channel-blockers. • A –blockers.
Pregnancy		• a-methyl dopa. • hydralazine.

2. Treatment of Malignant and Accelerated Hypertension:

- The goal of treatment is to lower the BP rapidly to arrest complications.
- The rapid reduction of BP should be avoided to avoid organ hypoperfusion.

By:-

- Bed rest is required, better in hospital.
- Treatment is usually started using a diuretic, B-blocker & vasodilator.

3. Treatment of Hypertensive Emergencies:

1. Bed rest is required, better in hospital (ICU).
2. Rapid reduction of blood pressure to approximately **100 mm Hg diastolic** within one hour using any of the rapidly acting drugs e.g.
 - **Na-nitroprusside**: 0.5-10 ug / kg / min IV infusion.
 - **Nifedipine**: 10 - 20 mg sublingually
 - **Dizoxide**: 50 - 100 mg / 5 - 10 min IV
 - **Hydralazine**: 10 - 20 mg / 30 min IV
 - **a-meth dopa**: 250 - 500 mg / 6 h IV
 - **IV frusemide** may be given to assist in control of BP and prevent fluid retention that may occur with the use of some antihypertensive drugs.
3. Venesection is rarely needed in resistant cases.
4. Following control of BP, oral antihypertensive treatment should be started usually with a diuretic, B-blocker & vasodilator.
5. Other lines of treatment according to the emergency.

e.g.**For Hypertensive encephalopathy:**

- Cerebral dehydrating measures: IV mannitol or concentrated glucose.
- Magnesium sulphate per-rectum.
- Anticonvulsants: IV diazepam & phenytoin

For subarachnoid & cerebral hemorrhage: (See Neurology)

For acute left ventricular failure: (See treatment of acute pulmonary)

For myocardial infarction & ischaemia: (See ischaemic heart disease).

For dissecting aortic aneurysm.

For renal failure:(See Nephrology).

Renal Artery Stenosis

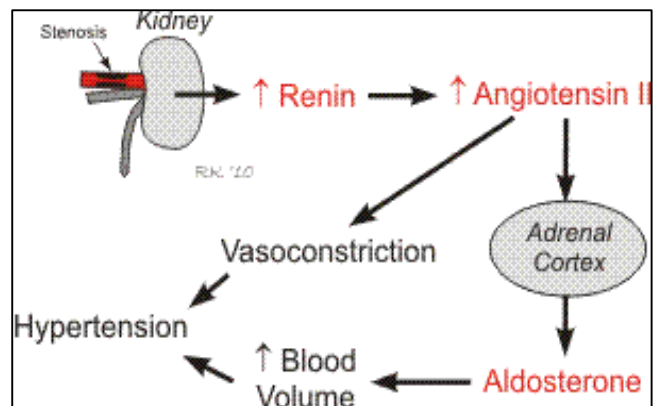
Aetiology:

- Atherosclerosis.
- Vasculitis e.g. polyarteritis nodosa.
- Renal artery thrombosis & embolism.

Clinical picture

Renal artery stenosis should be suspected in:

- Young Pt. less than 25 years.
- Of Sudden onset, severe and resistant HTN
- Bruit in the loin or lateral to the umbilicus.
- Evidence of other occlusive arterial disease.
- Hypertension not responding to medical treatment.



Investigations

- Isotopic scanning.
- Renal arteriography.

Treatment

- Control of hypertension.
- Surgical revascularization.
- Balloon dilatation, may be done for some patients.

Pulmonary hypertension

Definition:- Elevation of pulmonary arterial systolic pressure above 30 mm Hg and mean pressures above 20 mm Hg.

Causes

A. **Primary pulmonary hypertension (PPH):**

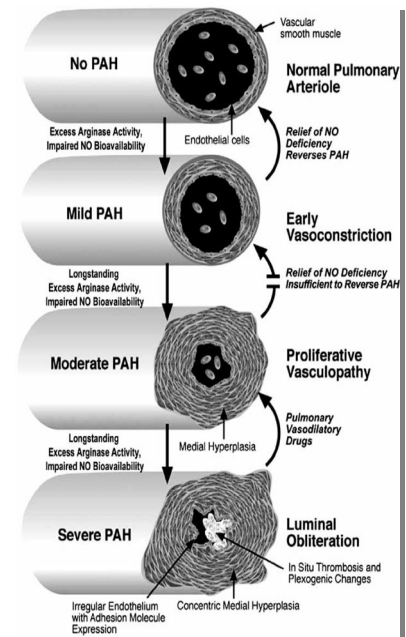
It occurs more commonly in middle-aged females with repeated pregnancies.

Theories:

1. **Endothelial dysfunction:** (Prostacyclin and nitrous oxide endothelin)
2. **Thrombosis in situ.**

B. **Secondary hypertension**

1. **Passive:** due to P congestion --- LSHF - MS.
2. **Hyperdynamic:** increased pulmonary blood flow ---
In congenital left to right shunt e.g. ASD, VSD & PDA.
3. **Vasoconstrictive:** It occurs in
 - Long standing passive pulmonary hypertension.
 - Long standing hyperdynamic pulmonary HTN
 - Hypoxia: e.g. RF (COPD, IPF, pulmonary A-V fistula and high altitudes)
4. **Obstructive (Obliterative)** of pulmonary arterioles:-
 - Long standing vasoconstrictive.
 - Diseases of the pulmonary arteries :- e.g.
 - Recurrent pulmonary emboli.
 - Pulmonary bilharziasis
 - Pulmonary vasculitis e.g. SLE, scleroderma.
 - Pulmonary thromboses in hypercoagulability e.g. sickle cell anaemia.
5. **Acute pulmonary hypertension:-** It occurs in :-
 - Massive pulmonary embolism.
 - Massive lung collapse.
 - Tension or bilateral pneumothorax.



Clinical picture

Symptoms

- Symptoms of low COP.
- Symptoms of right ventricular failure may occur lately.

Signs

General signs:

- Signs of low CO.
- Giant A wave (Or, **Prominent V wave in acute right ventricular failure (functional T.R.)**)
- Signs of right ventricular failure may occur lately.

Cardiac signs:**1. Precordial signs:**

- **Signs of pulmonary hypertension** (in the 2nd left space).
- Pulsations (palpable pulmonary component of S₂) (diastolic shock) & dullness.

2. Auscultation:

- **Heart sounds:** Accentuated P2 with closed splitting
- **Additional S:** Ejection click and S4 over tricuspid area
- **Murmur:**
 - ✓ Early diastolic murmur (Graham-Steell) due to functional **PR**.
 - ✓ Ejection systolic murmur due to relative **PS**.
 - ✓ Functional **TR**.

Investigation**1. Chest X-ray**

- Dilatation of pulmonary arteries in the hila with attenuation of periphery.
- Right atrial & ventricular enlargement.
- Features of the cause may be detected.

2. ECG

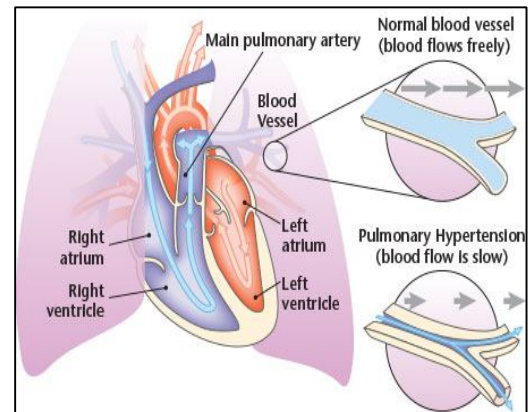
- P Pulmonale.
- Right ventricular enlargement.

3. Echocardiography & Doppler ultrasound:-

- Detection of right ventricular enlargement.
- Estimation of pulmonary arterial pressure.
- Features of the cause may be detected e.g. MS.

4. Cardiac catheterization:-

- As, Echo+ Measurement of pulmonary wedge pressure.

**Reversibility of pulmonary hypertension**

- Response to Vasodilator through Doppler or Cathetrization, or
- Ratio: pulmonary / systemic BP.

Treatment

- Treatment of the underlying causes.
- Direct vasodilators, Ca blockers & ACE inhibitors is usually of limited effect.
- Treatment of right sided heart failure.
- Anticoagulants for prophylaxis against secondary thromboembolism.
- Heart lung transplantation is the only radical treatment.

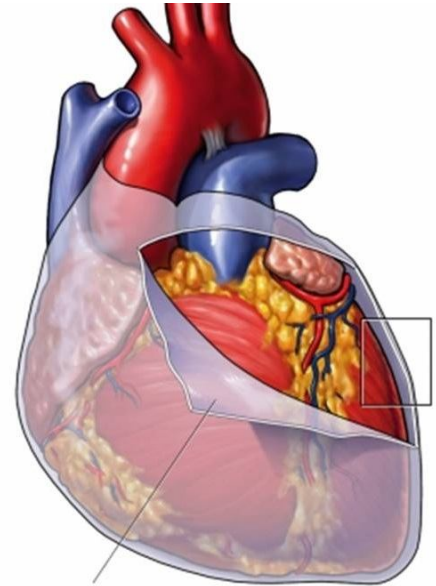
PERICARDIAL DISEASES

Dry pericarditis.	FAHM + Pain + Rub
Pericardial effusion.	HD changes:- S. congestion + Pressure manifestation + P
Constrictive pericarditis	
Adhesive pericarditis.	No HD changes

DRY PERICARDITIS

Aetiology

- I**nfection
 - Viral: Coxsackie B or Echovirus "most common".*
 - Bacterial: tuberculosis.*
 - Fungal.*
- I**diopathic: probably viral.
- I**nfarction: may occur as early or late complication.
- I**mmunological disease: SLE & rheumatoid arthritis.
- I**nfiltration: leukemia, L. carcinoma or mesothelioma.
- I**atrogenic: Hydralazine, procainamide.
- P**ost-infarction syndrome "Dressler's".
- P**ost-cardiotomy syndrome.
- R**heumatic fever: during carditis.
- R**enal failure. "metabolic: uremia"



Clinical picture

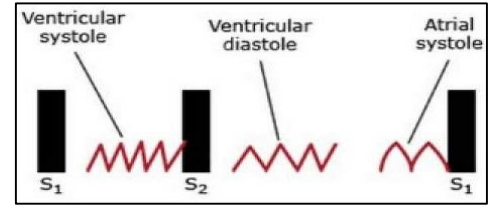
Symptoms

- Pain:**
 - * **Site:** retrosternal pain
 - * **Referred to:** shoulders & upper abdomen.
 - * **Severity:** it is severe
 - * **Type:** continuous & stitching or oppressive
 - * **Increases by:** breathing & rotation of trunk (no relation to exertion).
 - * **Relieved by:** sitting forewords.
 - * **Associated with:**
- General symptoms (**FAHM**):- fever, anorexia, headache & malaise.
- Symptoms of the cause.

Signs

- General signs:
 - Fever & sweating.*
 - Signs of the cause.*

- **Pericardial rub**:- High pitched superficial gritty or scratchy sound due to friction .
- * **Timing** :- One or more (1 systolic & 2 Diastolic)
- * **Site**: usually best heard in the **bare** area & base of heart.
- * **Character**: gritty or scratchy.
- * **Increases** on pressing the stethoscope against chest wall.



Complications:- Pericardial effusion

Investigations

ECG	- Raised S-T segment which is concave upwards & occurring in all ECG Leads - T waves: may be inverted or flat.
X- rsy	- No abnormality is detected.
Echo	- Small amount of pericardial fluid may be detected
Investigations for diagnosis of the cause	

Differential Diagnosis

1. Causes of acute chest pain.
2. Differential diagnosis of the cause.

Treatment

- Treatment of the cause.
- Anti-inflammatory & analgesic drugs e.g. **Indomethacin**.
- Resistant cases may require steroids.

PERICARDIAL EFFUSION

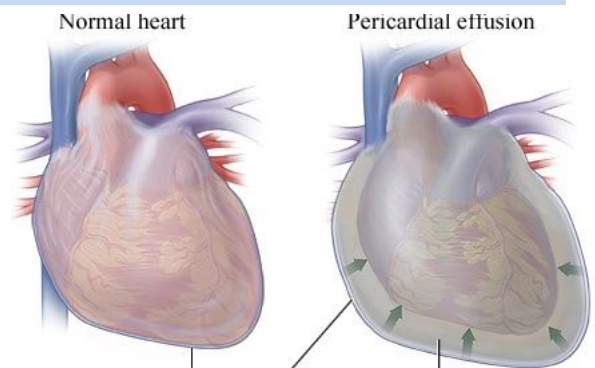
definition:- Accumulation of fluid in pericardial sac.

Aetiology

According to the nature of accumulated fluid:

A. **Seropericardium** (Accumulation of **Exudate**)

- **As dry pericarditis** especially:
 - ✓ It's due to ↑ capillary permeability.
 - ✓ T.B.: the most common cause.
 - ✓ Idiopathic (probably viral).
 - ✓ Rheumatic.



Special types

Haemorrhagic pericarditis is a severe form of (**serous effusion**) containing many **RBCs**:

- Malignant & leukaemic infiltrations - MI - Uraemia - T.B.

Suppurative pericarditis is a severe form of **serous effusion** containing many **pus cells** & occurring in pyogenic infection e.g. Staph. or Strept. infections.

B. Hydropericardium (Accumulation of **Transudate**)

- ✓ Right –sided heart failure.
- ✓ Hypoproteinaemia e.g. liver failure, nephritic syndrome.

C. Haemopericardium (Accumulation of **Blood**)

- ✓ Rupture of the heart, myocardial infarction, aneurysm & trauma
- ✓ Rupture of dissecting aneurysm of aorta.
- ✓ Rupture of coronaries: trauma during catheterization.
- ✓ Hemorrhagic blood disease e.g. purpura & Hemophilia.

D. Chylopericardium (Accumulation of **Lymph**).

- ✓ Obstruction of thorathic duct e.g. by tumors.
- ✓ Injury of thoracic duct by trauma.

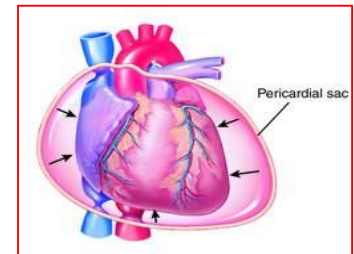
Haemodynamic:- Haemodynamic changes depend on: Amount of fluid - Rate of accumulation
Significant fluid (> 150 ml) ----- interferes with cardiac relaxation ----

Systemic congestion & Low CO.

Clinical Picture (S. congestio + L COP + Ps)

Symptoms

- Symptoms of S congestion.
- Symptoms of low CO.
- Precordial pain, dull aching pain may occur due to stretch of the parietal pericardium & may be referred to the shoulders.
- Pressure symptoms: may occur with massive effusion (Cough & dysphagia)
- Dyspnea: improved by sitting & leaning forwards (Prayer's position).
- Symptoms of the cause e.g. tuberculous toxemia.

**Signs****General Signs**

- Decubitus : may be Prayer's position.
- Pulsus paradoxus.
- Signs of low CO.
- Signs of systemic venous congestion:
- Neck veins
 - ✓ Congested Pulsating neck veins
 - ✓ Neck veins may be non-pulsating veins in severe cases.
 - ✓ Inspiratory filling (Kussmaul's sign): failure of the RA to accept the increased VR during inspiration.
 - ✓ Deep Y descent (Friedreich's sign) Diastolic collapse due to rapid emptying of the Atrium
- Enlarged tender liver.
- Ascites which may precede oedema of lower limbs (ascites precox)

Cardiac Signs**Inspection & palpation:**

- The apical pulsations are weak or absent.
- There may be precordial bulge in children.

Percussion:- (only here)

- Dullness outside the apex.
- Shifting dullness over the pulmonary area.
- Increased size of bare area with stony dullness
- Dullness to the right border of the sternum.
- Dullness over the left subscapular region.

Auscultation:

- Weak distant heart sounds.

Complications

- Cardiac tamponade (see later).
- Constrictive pericarditis.

Investigations**1. Lab Studies**

- **CBC:** evidence of infection or cytopenias
- **Cardiac enzymes** – To rule out myocardial ischemia.
- **for detection of the cause:-** RF, ANA, KFTs

2- Images:-**X-ray or screen**

- Enlarged flask-shaped cardiac shadow (**water-bottle heart**).
- Smooth cardiac borders (stenciled borders).
- Double contour of cardiac borders.
- Cardiophrenic angle is usually acute but in some cases it may be (obtuse) **Notch's** sign.
- Change of the shape of the cardiac shadow with change of position of the patient.
- Pulmonary oligoemia.
- Diminished pulsations of the heart under screen.

ECG

Low voltage & flat or inverted T wave.

Echo & Doppler

Highly diagnostic
Free space between the visceral and parietal pericardium
Early: accumulate posteriorly. **Severe:** diastolic collapse of the RA and RV. **Large effusions:** greater than 1 cm thick.
Small effusions: less than 1 cm and often localized.

Transesophageal echocardiography (TEE) is useful in loculated effusions

Ctscan	May detect as little as 50 cc of fluid.
MRI	Can detect as little as 30 cc of pericardial fluid. May be able to distinguish hemorrhagic and non hemorrhagic.
Catheter	Not required

3- Aspiration of effusion: (Pericardiocentesis):

May be needed for diagnosis of the cause by analysis of the pericardial fluid.

Features of exudates & transudate effusions

	Transudate	Exudate
Specific gravity	< 1.015	> 1.015
Total protein	< 3.0 mg /dL	> 3.0mg/dL
LDH	< 300 U/dL	> 300 U/dL
WBCs count	< 10,000	> 10,000
Protein fluid-to-serum ratio	< 0.5	> 0.5
LDH fluid-to-serum ratio	< 0.6	> 0.6
LDH >2/3 normal	Negative	Positive
Glucose fluid-to-serum ratio	> 1	< 1 (less reliable)

Sites of aspiration:

- Outside the cardiac apex.
- The bare-area of the heart.
- The angle between xiphoid process & left costal margin.

Complications:

- Ventricular rupture.
- Myocardial laceration or infection.
- Coronary artery laceration.
- Dysrhythmias.
- Pneumothorax.

Special tests:

- Viral cultures.
- **Gram stain**: for detection of organism - **Cultures**: infectious etiology.
- For TB: Adenosine deaminase, polymerase chain reaction (**PCR**).

Features of haemopericardium: The fluid is blood stained & contains many RBCs.

Features of chylopericardium:

The fluid is milky white, rich in fat and cleared on addition of ether
Stained orange with Sudan III.

4- Pericardial biopsy: May be needed for diagnosis of the cause e.g. TB.

5- Pericardioscopy.

Differential Diagnosis

1. Constrictive pericarditis.
2. Right-sided heart failure.
3. Restrictive cardiomyopathy.
4. Tricuspid stenosis.
5. Liver cirrhosis & nephritic syndrome.

Treatment

A. Medical

- Treatment of the cause: e.g. in tuberculous effusion
- Steroids may be added to reduce inflammation & prevent constrictive pericarditis.

Cardiac tamponade

It's a severest form of pericardial effusion.

Causes:- rapid massive causes of pericardial eff.
as: - ruptured myocardium, aneurysm, coronary
- traumatic - tumours

C/P: As Pericardial Eff. + Beck's triad

1- Low systolic BP .

2- Congested NV.

3- Weak HS (quite heart) .

Investigations & treatment : as pericardial effusion with emergent surgical drainage.

B. Pericardial aspiration: (Pericardiocentesis):

- In cardiac tamponade.
- May be used in suppurative effusion for drainage of pus & injection of antibiotics.
- May be used in (malignant effusion for injection of cytotoxic drugs).

C. Pericardial drainage:

1. **Subxiphoid pericardial window with pericardiostomy.**
2. **Thoracotomy** (Pleuropericardial window).
3. **Video-assisted thoracic surgery (VATS).**
4. **Balloon pericardotomy** by catheterization.

D. Pericardial sclerosis (Pericardiocentesis)

In **resistant cases** by using sclerosing agents (e.g., tetracycline, doxycycline, cisplatin)
Complications include intense pain, atrial arrhythmias and infection.

E. Pericardiectomy:

Indicated in cases of massive & recurrent effusions not responding to medical ttt.

Constrictive pericarditis "PICKS DISEASE"

Definition:- There is marked fibrous adhesion between the two layers of pericardium.

Aetiology

- The same as dry pericarditis with **exception of rheumatic fever.**
- **Tuberculosis** is the most common.

Haemodynamics:- as in pericardial effusion - **Calcification of pericardium is common**

Clinical Pictures**Symptoms**

- S. congestion.
- Low CO.
- Symptoms of the cause.

Signs**General Signs**

- Signs of low CO.
- Signs of S congestion (**as in pericardial effusion**).
- Pulsus paradoxus.
- AF: in 30% of cases.
- Signs of the cause.

Precordial examination:

- No cardiac enlargement.
- The apical pulsation are weak or absent
- Systolic retraction of the precordium.

Auscultation:

- Weak distant heart sounds.
- **S₃**: sudden halting of the relaxing ventricles by the pericardium (**pericardial Knock**).

Investigations

X- Ray	The heart is small & globular. Calcification of pericardium is diagnostic. Pulmonary oligoemia. Diminished pulsations of the heart under screen.
ECG	Low voltage & inverted T wave
Echo & Doppler	Pericardial thickening & may be calcification
Ctscan - MRI	Diagnostic
Catheter	Early diastolic dip followed by a plateau (square root sign) pressure tracings.

**Differential Diagnosis**

1. Pericardial effusion.
2. Right-sided heart failure.
3. Restrictive cardiomyopathy.
4. Tricuspid stenosis.
5. Liver cirrhosis & nephritic syndrome.

Treatment

- Pericardiectomy with treatment of the cause.
- Symptomatic treatment; e.g. for oedema & ascites.

ADHESIVE PERICARDITIS

Definition:- Fibrous adhesions between the pericardium and the surrounding mediastinal structures & chest wall

Aetiology:- It rarely occurs as a complication of rheumatic fever

Haemodynamic:- No HD

Clinical picture

- Clinical picture of associated rheumatic valvular lesions.
- Fixed apex: the apex does not change its site with change of position.
- **Broadbent's** sign: systolic retraction of the lower sternum & posterior intercostals spaces.

Investigation**Chest X-ray & fluoroscopy:**

- May be kinking of the oesophagus with cardiac contractions.
- May be cardiac enlargement due to associated valvular lesions.

Treatment

- Treatment of associated valvular lesions.

Cardiomyopathy

Definition:- group of disorders characterized by primary involvement of the myocardium.

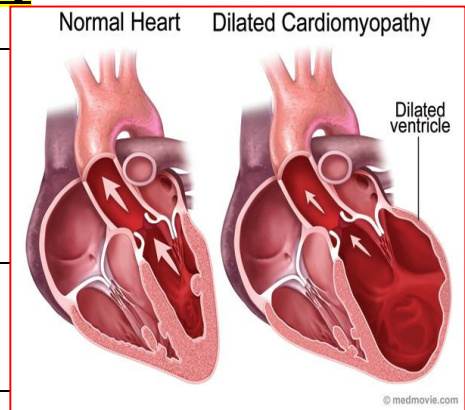
Types

1. Dilated (congestive) cardiomyopathy.
2. Hypertrophic cardiomyopathy (HOCM, IHSS).
3. Restrictive (non-dilated, non-hypertrophic) cardiomyopathy.

A. Dilated cardiomyopathy

Aetiology:-

4I	<u>I</u> nfectious: Viral especially coxsackie B virus & Chaga's disease. <u>I</u> mmunological e.g. SLE. <u>I</u> diopathic (may be Familial). <u>I</u> schemic (diffuse myocardial ischaemia).
T	<u>T</u> oxic: Alcohol – Anthracyclines.
E	<u>E</u> ndocrinal: Myxoedema – DM – Acromegaly
N	<u>N</u> eurologic: Duchienne's My, Myotonia & Friedreich's A



Haemodynamics:- (LV or both)

- **Systolic Dysfunction** - Decreased Pump.
- **Dilatation.**

Clinical picture

Symptoms	- Low CO. - Pulmonary congestion. - Systemic congestion.				
General	- Low CO. - S. congestion.				
Local	<table border="1"> <tr> <td>Precordial</td><td>- Biventricular enlargement. - Weak apex.</td></tr> <tr> <td>Auscultation</td><td>- Weak heart sounds. - - Murmurs of functional mitral & tricuspid incompetence</td></tr> </table>	Precordial	- Biventricular enlargement. - Weak apex.	Auscultation	- Weak heart sounds. - - Murmurs of functional mitral & tricuspid incompetence
Precordial	- Biventricular enlargement. - Weak apex.				
Auscultation	- Weak heart sounds. - - Murmurs of functional mitral & tricuspid incompetence				

Complications

- Arrhythmias: e.g. AF & ventricular tachycardia.
- Embolism: systemic & pulmonary.
- Sudden death.

Investigations

X- Ray	Cardiac enlargement involving all chambers. Pulmonary congestion.
ECG	- Low voltage.- Depressed ST segment.- Flat or inverted T wave.
Echo & Doppler	Dilated ventricles with no hypertrophy - Decreased contractility. Mitral & tricuspid incompetence.

Treatment

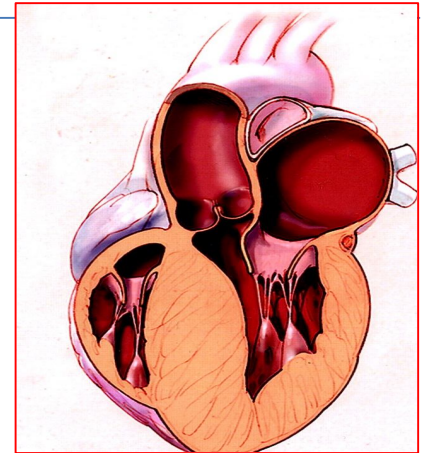
- Treatment of HF (may be refractory).
- Treatment of complications.
- Treatment of the cause if possible.

HYPERTROPHIC CARDIOMYOPATHY**Causes:**

- Familial: autosomal dominant.
- Idiopathic. (**IHSS**)
- Pheochromocytoma.
- Young athletes:- may cause sudden death

Hemodynamics: (LV)

- **Diastolic Dysfunction** (i.e. compliance) ---- P. congestion & decreased pump.
- Obstruction of outflow (**low CO**) -
- **Bernhiem effect**:- The septum may encroach on RV cavity ---- mild impairment of filling & giant A wave.

**Clinical picture**

Symptoms	-Pulmonary congestion. - Low CO.	
General	- Signs of low CO - Pulse: jerky or bisferiens pulse - Giant A wave .	
Local	Precordial	- LV enlargement - Double apex
	Auscultation	- Murmur of MR may be present. - Obstruction of outflow murmur (dynamic)**.

**Midsystolic murmur heard along left sternal border increases with standing & decreases with squatting. (Dynamic). + No Ejection click

Complications

- Arrhythmias, especially (AF).
- Sudden death.

Investigations:

X- Ray	- Left ventricular hypertrophy - Pulmonary congestion.
ECG	- Left ventricular enlargement. - Q waves in left chest leads (V5 & V6).
Echo & Doppler	- Hypertrophy of the left ventricle with the (septal wall thickness). - Usually exceeding free wall thickness (diagnostic).

Differential diagnosis

Aortic stenosis.

Treatment

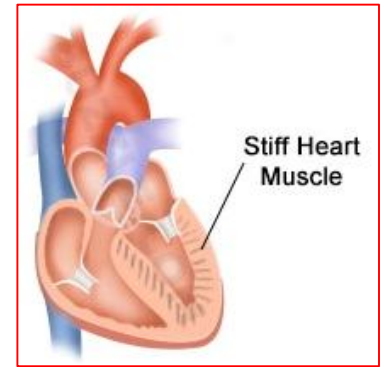
- Treatment of heart failure with avoidance of positive inotropic.
- **B-blockers (e.g. Propranolol) or Calcium channel blockers (e.g. verapamil).**
- Surgical treatment: resection of septum.

B. RESTRICTIVE CARDIOMYOPATHY**Causes**

- Amyloidosis.
- Sarcoidosis.
- Idiopathic.
- Hypereosinophilic syndrome.
- Haemochromatosis.
- Scleroderma.
- Endomyocardial fibrosis.

Haemodynamics (RV)

- Decreased **diastolic compliance** ----- S. congestion.
- No hypertrophy or dilatation (later).

Clinical picture

Symptoms	-S congestion. - Low CO (Late).	
General	- Signs of S. congestion* - low CO (Late)	
Local	Precordial	- RV enlargement (late).
	Auscultation	- Murmur of TR & MR (Papillary Ms fibrosis)

*Congested pulsating neck veins with inspiratory filling
 Deep X & Y descents (As, constrictive pericarditis).

Investigations

X- Ray	- RV ++ (late)
ECG	- Low voltage
Echo & Doppler	Decreased ventricular cavity

Differential diagnosis

	Restrictive cardiomyopathy	Constrictive pericarditis
Cardiomegaly	Late	Absent
Calcification	Absent	May be present
CT & MRI		Diagnostic
Endomyocardial biopsy	Diagnostic	
Exploratory thoracotomy	Diagnostic	Diagnostic

Treatment

- Treatment of heart failure which is usually refractory to therapy.
- Treatment of the cause if possible.

Comparison

	Dilated	Hypertrophic	Restrictive
Causes	4 I + TEN	Familial – idopathic. Pheochromocytoma	Infiltration
Haemodynamics	LV or Both Systolic Dysfunction	LV Diastolic Dysfunction Obstruction of outflow Bernhiem	RV Diastolic Dysfunction
Symptoms	Low CO. P. congestion. S. congestion.	P. congestion. Low CO	S. congestion. ± Low CO
General signs	Low CO. S. congestion.	Jerky or bisferiens pulse. Low CO. Giant A wave.	S. congestion. Low CO.
Precordial	LV & RV ++ (weak apex).	LV hypertrophy	RV enlargement.
Auscultation	Weak heart sounds. Functional MR & TR.	Midsystolic dynamic. Murmur of MR.	Murmur of TR & MR
Complications	Arrhythmias. Sudden death or embolization.	Arrhythmias. Sudden death	
D.D.		Aortic stenosis	Constrictive pericarditis
Chest X-ray	All chamber +++ P. congestion.	LV hypertrophy. P. congestion.	RV enlargement.
ECG	Low voltage. Depressed ST Flat or inverted T	LV enlargement. Q waves (V5 & V6).	Low voltage.
Echo	All chambers dilated. Decreased contractility. MR & TR.	Hypertrophy of LV . Septal wall thickness.	Decreased ventricular cavity.

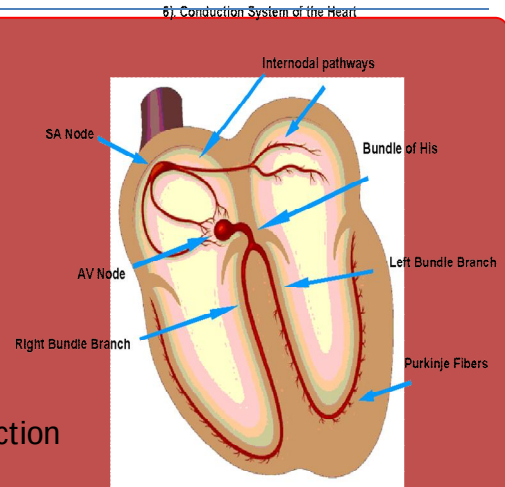
Dysarrhythmias

Definition:- Any disturbance of Rate, Rhythm or Atrio-ventricular relation.

Physiological Aspects

Normally, the heart rate is 60 - 100 beats/minute - Regular.

- **The SAN (Sino-atrial node):**
 - ✓ It is the normal pacemaker.
 - ✓ At the junction of the SVC and the right atrium.
 - ✓ Sympathetic accelerates, while vagus slows the SAN.
 - ✓ Impulse transmission from the S.A. node through the 3 internuclear bundles.
- **A-Vnode (Atrioventricular node):**
 - ✓ On the atrial side of the annulus fibrosus.
 - ✓ Sympathetic accelerates while vagus slows the conduction in A-VN
 - ✓ Represented by the P-R interval.
 - ✓ No retrograde conduction.
 - ✓ The ventricles are supplied by Sympathetic fibers but not the vagus (vagus escape phenomenon).
- **The right bundle branch** continues down the right ventricular endocardial surface to reach the anterior and apical muscle of the right ventricle.
- **The left bundle branch** crosses the summit of the muscular interventricular septum to emerge on the left side of the heart. The left bundle divides into a left posterior fascicle and a left anterior fascicle.



Classification

A. Aetiological classifications

1. Abnormal automaticity

a. S.A.N.	• Sinus tachycardia. • Sinus bradycardia. • Sinus dysarrhythmia. - Sinus arrest.
b. A.V.N.	• Junctional (nodal) premature beats. • Junctional (nodal) rhythm. • Paroxysmal junctional (nodal) tachycardia.
c. Atrial	• Atrial premature beats. • Paroxysmal atrial tachycardia. • Atrial flutter. • Atrial fibrillation. (AF)
d. Ventricular	• Ventricular premature beats. • Ventricular tachycardia (VT). • Ventricular fibrillation (VF)

2. Abnormal conductivity

A- S.A. block.	
B- A.V. block:	• 1st grade: P-R interval > 0.22 sec. • 2nd grade: Mobitz type one (Wenckenbach phenomenon) & Mobitz type two. • 3rd grade: complete heart block.
C- Bundle block:	• Right bundle branch block (RBBB). • Left bundle branch block (LBBB). • Left anterior hemiblock (LAHB). • Left posterior hemiblock (LPHB).

B. Rhythmic classifications

	Regular	Irregular
Tachy	• Sinus tachycardia. • Paroxysmal supraventricular tachycardia. • Atrial flutter. • Ventricular tachycardia.	• AF. • Sinus tachycardia with premature beats. • Paroxysmal supraventricular tachycardia with variable heart block. • Atrial flutter with variable heart block.
Brady	• Sinus bradycardia. • 1st heart block. • Junctional (nodal) rhythm. • Partial fixed heart block (2:1, 3:1, 4:1, etc.). • Complete heart block.	• Slow partial variable heart block. • Sinus bradycardia with premature beats. • Sinus bradycardia with respiratory sinus dysarrhythmia.

In any cases of Dysrhythmia

Definition	- Rhythm	Rate	Origin of beat.....
	+ Special features.....		

Aetiology:-

A- General causes:	1. Simple causes (stress, sadness, smoking , coffee and tea) 2. Thyrotoxicosis () 3. Drugs e.g. digitalis, sympathomimetics. 4. Electrolyte disturbances: (eg. Hyperkalaemia).
B- Cardiac causes:	1. Ischaemic heart disease especially myocardial infarction. 2. Myocarditis & cardiomyopathies. 3. Rheumatic & congenital heart diseases.
C- Specific causes:	1. <u>Heart block</u>: • Drugs e.g. digitalis & B-blockers. • Fibrosis & calcification of the AV node & bundle. 2. <u>AF</u>: • MS • Constrictive pericarditis. • Chest diseases e.g. pulmonary embolism, COPD. • Pre-excitation syndromes e.g. WPW syndrome. • Lone AF.

N.B. Sinus dysrhythmia (3p:- Physiological - Pathological - Pharmacological)

Diagnosis:-**A- Clinical Picture :**1. Symptoms:

Tachy	Brady
- May be asymptomatic. Palpitation:- onset.....offset.....duration..... and number of recurrence - Symptoms of low CO. - Precipitation of HF in susceptible patients.	
Thromboembolism especially in AF.	
Angina - Ventricular arrhythmia may result in sudden death.	2nd & 3rd grades heart block may result in Syncope or Sudden death.

2. Signs:**Arterial Pulse (4Rs):**

- Rhythm regular ----- **except** (AF-Extrasystole)
- Rate.....
- Response to -----
 - Carotid** stimulation:- slow down HR in all tachy **except Ventricular ??**
 - Excises**:- for brady group

4. Respiratory sinus rhythm: It is the acceleration of the heart rate during inspiration (due to increased venous return) only **positive in sinus rhythm**.

Neck veins (4)

1. Rhythm
2. Rate
3. Waves (A & V waves).....
4. A & V relationship

Heart sound (S1): ($S1 \propto HR$).....

Tachycardia	Accentuated
Bradycardia	Muffled
A-V dissociation	Cannon (occasional)
Junctional	Cannon (regular)
Irregular	Variable

Cannon waves & Cannon sounds:

Simultaneous contraction of atria & ventricles results in marked systolic expansion of neck vein (Cannon waves) & very loud first heart sound (cannon sounds).

B- Investigations

1. **ECG:** (Rest ECG or Holter monitoring) Comment on **(4)**:
 - a. P wave: rhythm, rate & morphology.....
 - b. QRS complex: rhythm, rate & morphology.....
 - c. P-R interval
 - d. P and QRs relationship.....

P wave	Single P	Sinus arrhythmias .
	Bizarre, inverted or absent	Atrial arrhythmias .
	Multiple	Atrial flutter.
	Absent or fibrillation	Atrial fibrillation.
P-R interval	Short	Tachycardia (mainly WPW syndrome)
	Prolonged	HB (mainly 1st degree)
QRS complex	Bizarre	V. tachy - 3rd HB
	Wide	WPW

2. **Electrophysiological studies:**
3. **Diagnosis of the cause of dysrhythmia:**

Cardiac investigations: e.g. Chest X-ray, ECG, echo & cardiac Catheterization.

Laboratory investigations: e.g. serum K & thyroid function tests.

Treatment:- According to the type of dysrhythmia

SINUS Tachycardia

Definition:-

It a condition in which the SAN discharges at a high rate (**> 100 / min**) & regular rhythm.

Aetiology (3Ps):

1. **P**hysiological: - Exercise, emotional stress, pregnancy & infancy.
2. **P**athological: Fever, hyperdynamic circulation, heart failure, shock & myocarditis.
3. **P**harmacological: e.g. adrenaline, atropine, thyroxine, nifedipine.

Clinical picture

Symptoms

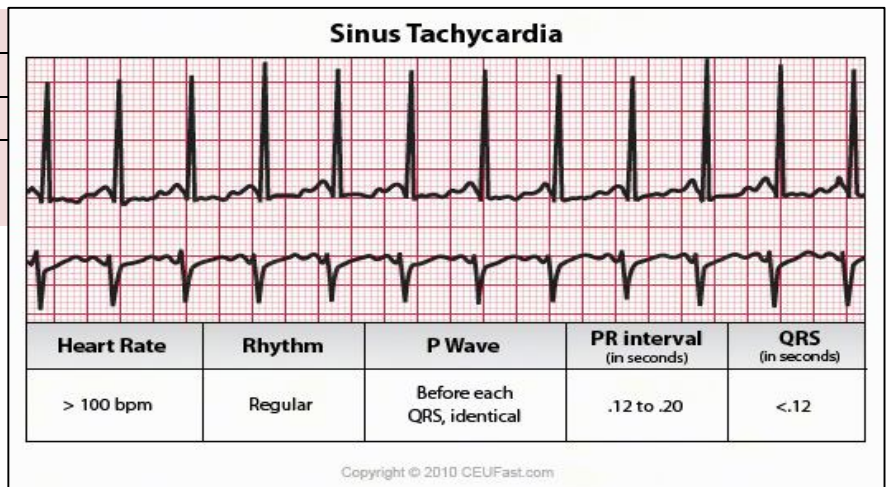
1. May be asymptomatic.
2. Palpitation: rapid regular palpitation of gradual onset.
3. Symptoms of low CO may occur in severe cases.
4. May precipitate angina & heart failure in susceptible patients.

Signs

Pulse	Rhythm	Regular
	Rate	100-180
	Response to	Carotid:- Gradual slowing then gradual return to original HR
	Res. sinus rhythm	Positive
Neck V	Rhythm	Regular
	Rate	100-180
	Wave	Normal
Auscultation	S1	Accentuated

Investigation:-

ECG	Rhythm	Regular
	Rate	100-180
	P-R	Short
	Waves	Each P followed by a QRS



Treatment

1. Treatment of the cause.
2. Sedation may be needed.
3. B-blockers may be given if there is no contraindication e.g. propranolol.

Paroxysmal supra ventricular tachycardia (PSVT)

Definition:-

- It is a dysrhythmia originating from the **atrium** (atrial tachycardia) or the **AVN** (junctional tachycardia).
- It is characterized by being **regular**, **rapid**, of sudden onset, sudden offset, short duration and tendency for recurrence (**Paroxysmal**).

Aetiology (as usual + WPW)

Cardiac causes

- Ischaemic heart disease especially myocardial infarction.
- Myocarditis & cardiomyopathies.
- Rheumatic & congenital heart diseases.

General causes

- Simple causes (stress, sadness, smoking, coffee and tea).
- Thyrotoxicosis.
- Drugs e.g. digitalis, sympathomimetics.
- Electrolyte disturbances: (e.g Hyperkalaemia)

Specific causes:

- Pre-excitation syndromes e.g. **WPW** syndrome.

May Occur in apparently normal individuals.

Clinical Picture

Symptoms

- Never to be asymptomatic. Paroxysmal**
- Palpitation: rapid regular palpitation of sudden onset, sudden offset, short duration and tendency for recurrence.
- Symptoms of low CO may occur in severe cases.
- May precipitate angina & heart failure in susceptible patients.

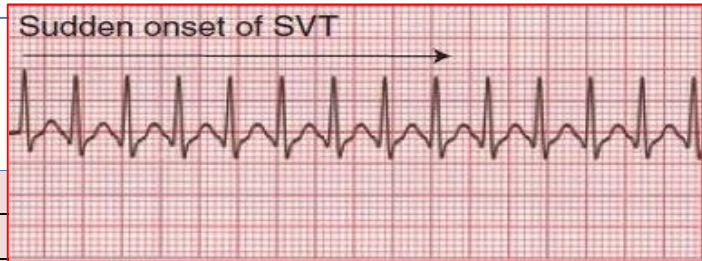
Signs

Pulse

Pulse	Rhythm	Regular
	Rate	150-250
	Response to	Carotid :- reversion to initial HR may occur in some patients
	Res. sinus rhythm	negative
Neck V	Rhythm	Regular - 150-250
	Rate	*Atrial tachycardia: normal waves with the same rate of pulse
	Wave	* Junctional tachycardia: regular cannon with the same rate of pulse
Auscultation	S1	* Atrial tachycardia: accentuated S ₁ .
		* Junctional tachycardia: regular cannon, sounds.

Investigations:-

ECG	Rhythm	Regular
	Rate	150-250
	Waves	QRS :- normal morphology P wave :- - Deformed in <u>Atrial tachycardia</u> - Inverted or absent in <u>Junctional tachycardia</u>



	Atrial tachycardia	Junctional tachycardia
Neck vein		
Rhythm.	Regular	
Rate	With the same rate of pulse (150-250/min)	
Waves	Normal	Cannon
Auscultation		
S1	Accentuated	Cannon
ECG		
Rhythm:	Regular	
Rate	With the same rate of pulse (150-250/min)	
QRS	Normal morphology	
P Wave	Deformed	Inverted or absent

Treatment**A. During the attacks:**

1. Vagal stimulation: using carotid sinus massage (or less preferably ocular compression or induction of vomiting).
2. If there is no response: Verapamil 5-20mg IV or Digitalis IV or Propranolol IV.
3. If there is no response and the patient is haemodynamically unstable (e.g. presence of hypotension or heart failure): DC is indicated.

B. In between the attacks:

1. Treatment of the underlying causes.
2. Maintenance therapy with antiarrhythmic drugs e.g. verapamil, digitalis, Propranolol or quinidine orally.
3. In (resistant case) of pre-excitation syndromes: division of the accessory pathway is indicated.

Atrial flutter

Definition

It is a condition in which the **atria** discharge at a **regular** rapid rate (**250 - 350/min**)

- Due to the physiological block at the AVN only 1/2, 1/3, 1/4, etc of the atrial impulses are transmitted to the ventricles (**A rate > V rate**)

Aetiology (As, usual + WPW)

Cardiac causes:

1. Ischaemic heart disease especially myocardial infarction.
2. Myocarditis & cardiomyopathies.
3. Rheumatic & congenital heart diseases.

General causes:

1. Simple causes (stress, sadness, smoking, coffee and tea)
2. Thyrotoxicosis.
3. Drugs e.g. digitals, sympathomimetics.
4. Electrolyte disturbances: (eg, Hyperkalaemia).

Specific causes:

- Pre-excitation syndromes e.g. **WPW** syndrome.

Clinical Picture

Symptoms:

1. Palpitation: rapid regular palpitation of sudden onset, sudden offset, short duration and tendency for recurrence.
2. Symptoms of low CO may occur in severe cases.
3. May precipitate angina & heart failure in susceptible patients

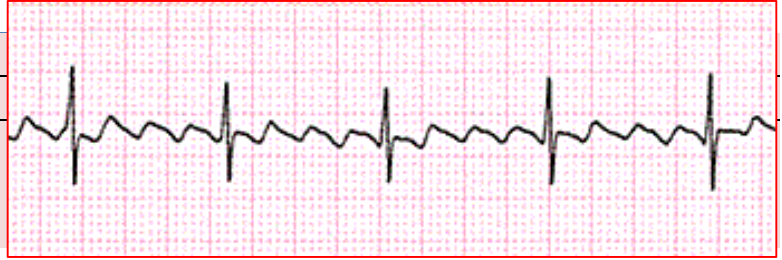
N.B. The condition may changes to sinus spontaneously, or progress to AF.

Signs

Pulse	Rhythm	Regular
	Rate	120-180 depending on the degree of A-V conduction
	Response to	Carotid:- HR decreases in a mathematical fashion (e.g. 150-100- 75) On release:- the rate returns to its initial HR by the same fashion
	Res. sinus rhythm	Negative
Neck V	Rhythm	Regular
	Rate	120-180
	Wave	Multiple A waves before each V according to A/V ratio.
Auscultation	S1	Accentuated

Investigations

ECG	Rhythm	Regular
	Rate	120-180
	Waves	Multiple P waves before each QRS (Saw-tooth appearance)



Treatment

1. **In haemodynamically unstable patient** (e.g. presence of hypotension or heart failure): Cardioversion by Overdrive pacing is indicated.
2. **Haemodynamically stable patient:** Verapamil 5 - 20 mg IV or Propranolol IV.
3. **Digitalis:** could be given to slow the ventricular rate by decreasing conduction in the AVN. After stoppage of digitalis atrial flutter is converted to AF or sinus rhythm may be restored.
4. **For prevention of recurrence:** class I antiarrhythmic (e.g. quinidine) alone or with digitalis is usually given.

Atrial Flutter				
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 220-430 bpm V: <300 bpm	Regular or variable	Sawtoothed appearance	N/A	<.12

Ventricular tachycardia (Paroxysmal)

Definition

It is a dysrhythmia originating from the ventricles & characterized by being **regular** rapid, of sudden onset, sudden offset, short duration and tendency for recurrence (**Paroxysmal**)

- The ventricles will be controlled by the (ectopic focus) while the atria controlled by the SAN (A-V dissociation).

Aetiology

Cardiac causes:

1. Ischaemic heart disease especially myocardial infarction.
2. Myocarditis & cardiomyopathies.
3. Rheumatic & congenital heart diseases.

General causes:

1. Simple causes (stress, sadness, smoking, coffee and tea).
2. Thyrotoxicosis.
3. Drugs e.g. digitalis, sympathomimetics.
4. Electrolyte disturbances: (e.g. Hyperkalaemia).

Clinical Picture

Symptoms

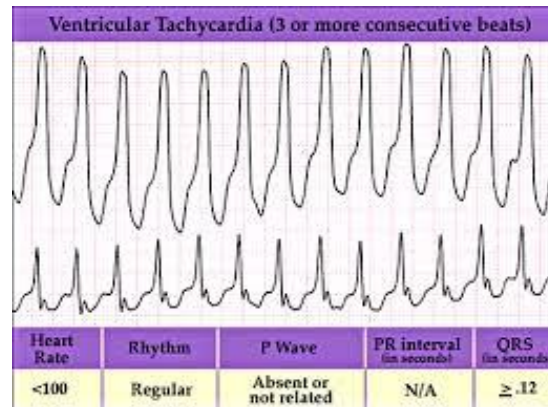
1. Never to be asymptomatic.
2. Palpitation: rapid regular palpitation of sudden onset, sudden offset, short duration and tendency for recurrence.
3. Symptoms of low CO may occur in severe cases.
4. May precipitate angina & heart failure in susceptible patients.
5. **Sudden death** (if converted to ventricular fibrillation).

Signs

Pulse	Rhythm	Regular
	Rate	100-220
	Response to Carotid:-	no effect (Vagal escape phenomenon)
	Res. sinus rhythm	Negative
Neck V	Rhythm	Regular
	Wave	Occasional Cannon waves Rate of A wave: 60-100/min - No relation between A wave and V wave.
Auscultation	S1	Variable with occasional cannon sound

Investigations

ECG	Rhythm	Regular
	Rate	100-220
	Waves	QRS complex: of rapid regular rate and deformed (bizarre) no relationship between P & QRS complex



Treatment

A. During the attack

1. If the patient is hemodynamically stable:

- Amiodarone or Procainamide or amiodarone: IV.
- Lidocaine: initial bolus of 2 mg / kg IV, followed by infusion of 1 – 4 mg / min.
(it was the drug of choice for a long time in all cases but now, it is only considered in peri-infarction V. tach - adenosin is CI)
- Bretylium: IV (in resistant cases).

2. If the patient is hemodynamically unstable:

DC cardioversion immediately, followed by Pharmacological ttt.

B. Prevention of a future attack:

1. Maintenance therapy:

Drugs: (oral), class one or class three anti-arrhythmic drugs.

- Intervention:** Ablation of focus: **Catheter** (radiofrequency energy) or **surgery**.
Implantable Cardioverter Defibrillator (ICD): "Anti- tachycardia pacing".

3. Treatment of the cause.

Rapid regular

	Sinus tachycardia	PSVT		Atrial flutter	PVT
Definition					
Rhythm	Regular				
Rate	> 100 / min	150 – 250	250 – 350	100 – 220	
Origin of beat	SAN	Atrial or nodal	Atrial	Ventricular	
Special features			Reductive conductivity	A-V dissociation	
Aetiology	3Ps	As usual + WBW			As usual
Clinical pictures					
Symptoms	As usual	As usual (never asymptomatic).	As usual	As usual (never asympt).	
Signs					
Arterial pulse (4Rs)					
Rhythm	Regular				
Rate	> 100 / min	150 – 250 / min	250 – 350	10 – 220	
Response to Carotid	Gradual slowing		Mathematical reduction in HR	No effect	
R. S. rhythm	Positive	Negative	Negative	Negative	
Neck vein (4)					
Rhythm	Regular				
Rate	> 100 / min	Atrial	Nodal		
Waves	Normal	Normal	Reg. cannon	A > V	
A & V relationship	Normal	As pulse		Occas. cannon	
Heart sounds S ₁	Accentuated	Accent	Reg. cannon	Accentuated	
ECG					
P wave	Normal	Deformed	Inverted	Multiple small (saw tooth)	
QRS	Normal	normal		Deformed (50%)	
P-R interval	Normal	Normal		Normal	
P and QRS relationship	Each P followed by QRS	Each P followed by QRS	P > QRS	No relation	

Atrial fibrillation (AF)

Definition

It is a condition in which the atria discharge impulses irregularly at a high rate of 400-600/min.

Due to the physiological block in the AVN, the ventricular rate is less than the atrial rate.

Hemodynamic Effects

Atrial fibrillation is associated with the loss of the atrial contribution to ventricular filling. This may result in a decrease in ventricular stroke volume of up to 20%.

Aetiology

Cardiac causes:-

1. Ischaemic heart disease especially myocardial infarction.
2. Myocarditis & cardiomyopathies.
3. Rheumatic & congenital heart diseases.

General causes:

1. Simple causes (stress, sadness, smoking, coffee and tea).
2. Thyrotoxicosis.
3. Drugs e.g. digitalis, sympathomimetics.
4. Electrolyte disturbances: (eg. Hyperkalaemia).

Specific causes:

1. MS
2. Constrictive pericarditis.
3. Chest diseases e.g. pulmonary embolism, COPD.
4. Pre-excitation syndromes e.g. **WPW** syndrome.
5. Lone AF*.

The term "**lone atrial fibrillation**" describes atrial fibrillation in the absence of demonstrable underlying cardiac disease or a history of hypertension. It may be due to fibrotic areas in the atrium that predisposes patients to arrhythmia.

Clinical Picture

Symptoms:

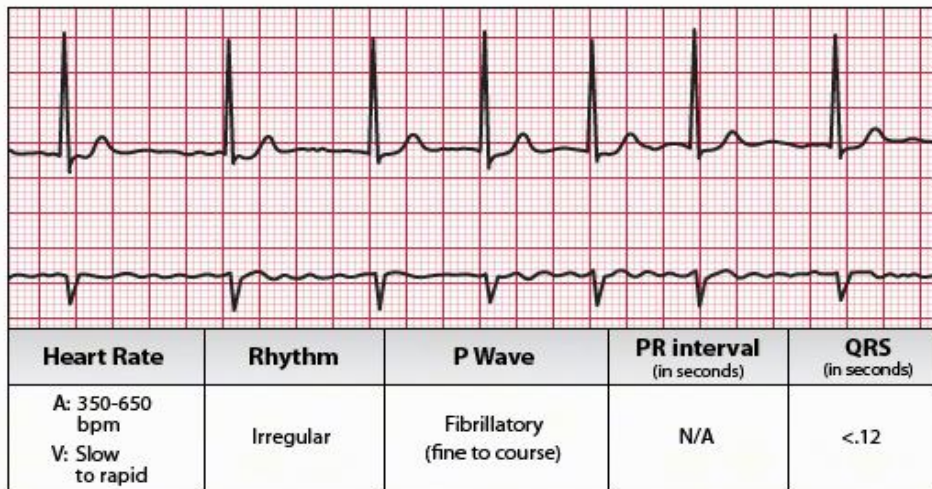
1. Palpitation: rapid irregular, may be paroxysmal or persistent.
2. Symptoms of low CO.
3. Precipitation of angina & heart failure in susceptible patients.
4. May cause atrial thrombosis & embolizations.

Signs

Pulse	Rhythm	Marked irregularity
	Rate	Usually rapid e.g. 100-150/min. Slow AF :- heart block - patients receiving digitalis or propranolol - lone AF*)
	Response to	Carotid :- slowing of pulse due to decreased AV conduction. Exercise :- increased irregularity due to increased AV conduction
	Res. sinus rhythm	Negative
	Pulse deficit : more than 10 beats/min	
Neck V	Rhythm	Marked irregularity
	Rate	Usually rapid except
	Wave	absent A wave with prominent V (Systolic expansion)
Auscultation	S1	Marked irregularity & variability

Investigations

ECG	Rhythm	markedly irregular rhythm
	Rate	Usually rapid except
	Waves	Absent P waves which are replaced by irregular vibrations (fibrillation waves) QRS complexes are of normal shape & markedly irregular rhythm

Atrial Fibrillation**Types of AF**

- 1. Newly Diagnosed Atrial Fibrillation**
- 2. Recurrent Paroxysmal Atrial Fibrillation.**
- 3. Persistent Atrial Fibrillation:**
Once an episode of atrial fibrillation has lasted more than **seven days**, spontaneous conversion is rare and the condition can be defined as persistent.
- 4. Drug-Refractory Atrial Fibrillation:**

Treatment of AF either (Rate control or Rhythm control)**A. Treatment of the cause.****B. Cardioversion to sinus****Rhythm control****1. Indicated in the following conditions:**

- Recent AE (less than 12 months duration).
- No history of embolism.
- No marked enlargement of atria.

2. Types:

- Electrical & Chemical by quinidine.

	Electrical	Chemical
<i>should be preceded by</i>	Anticoagulation	
<i>Procedure</i>	Discontinuation of digitalis <u>Electrical cardioversion</u> (of choice)	Digitalis <u>Quinidine:</u> 1. 1 tablet (0.2 gm) as test for hypersensitivity. 2. (2 tab / 2 h) until sinu-version or 5 doses. 3. IF failed:- larger doses could be used in the second day. 4. In toxicity, quinidine should be stopped. 5. Maintenance dose:- half dose of sinu-version dose.

C. Control of ventricular rate-(Reversion to sinus rhythm is not indicated)

- Long standing AF.
- History of embolism.
- Marked enlargement of atrium.

Rate control

TTT: Digitalis, verapamil or propranolol are indicated to slow the ventricular rate.

D. Anticoagulation: is indicated in following condition:

- Before reversion to sinus rhythm
- Previous embolizations.
- Valvular diseases as MS.

Premature beats (Extrasystoles)

Definition

One of the irregular dysrhythmias due to ectopic impulses arising from the atria, AVN or ventricles - causing premature beats followed by compensatory pause.

Aetiology

May occur in normal person: e.g. due to anxiety, excessive smoking, coffee & tea.

Cardiac causes:

1. Ischaemic heart disease especially myocardial infarction.
2. Myocarditis & cardiomyopathies.
3. Rheumatic & congenital heart diseases.

General causes:

1. Simple causes (stress, sadness, smoking, coffee and tea)
2. Thyrotoxicosis
3. Drugs e.g. digitalis, sympathomimetics.
4. Electrolyte disturbances: (eg. Hyperkalaemia).

Clinical Picture

Symptoms

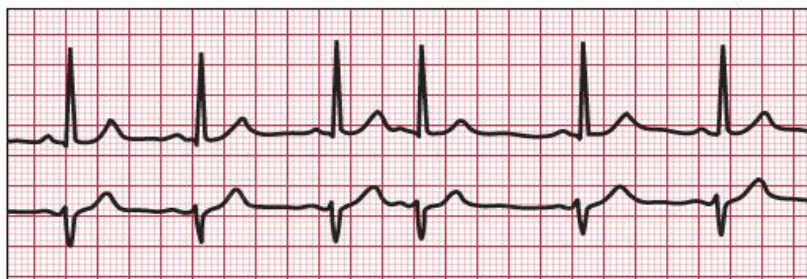
- May be asymptomatic.
- Irregular palpitation. (Described by the patient as an occasional filling of missed or repeated beats).

Signs

Pulse	Rhythm	Mild (occasional) irregularity
	Rate	Sinus
	Response to	Exercise:- decrease irregularity due to shortening of diastole
	Res. sinus rhythm	Positive
	Pulse deficit: Less than 10 beats/min	
Neck V	Rhythm	Occasional irregularity
	Rate	Sinus
	Wave	Normal waves
Auscultation	S1	normal sounds with Occasional irregularity

Investigations

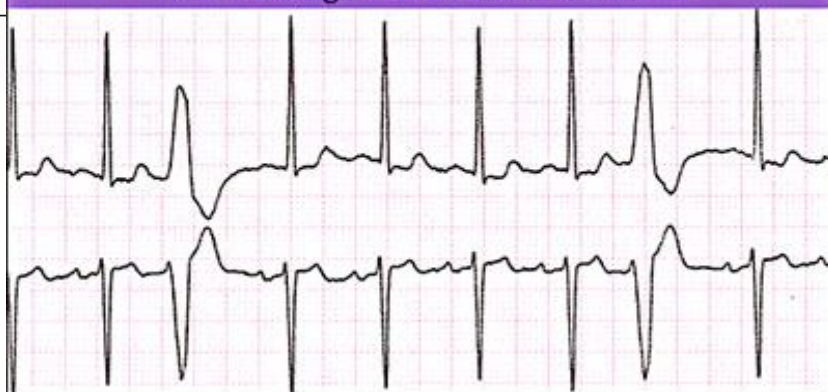
ECG	Rhythm	Mild irregular rhythm
	Rate	Sinus
	Waves	Prematurely beat followed by a compensatory pause Supraventricular: inverted or absent P wave followed by normal QRS complex. Ventricular: deformed QRS not preceded by P wave.

Premature Atrial Contraction • Isolated PAC's: Occur Single

Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
N/A	Irregular	Premature & abnormal or hidden		

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Unifocal PVC's: identical shapes
Note: A single PVC is labeled isolated

**Differential diagnosis of irregular tachycardia**

	Extrasystole (with S. tachycardia)	AF
Pulse		
Rhythm	Occasional irregularity	Marked irregularity
Rate	According to sinus	Usually rapid e.g. 100 – 150 / min
Carotid massage		+ Slowing of pulse
Exercise	Decrease irregularity	Increased irregularity
Pulse deficit	< 10 beats / min	> 10 beats / min
Respiratory sinus rhythm	Positive	Negative
Neck vein		
A wave	Normal with occasional irregularity	Absent
V wave		Systolic expansion
Heart sounds		
	Normal with occasional irregularity	Variability
ECG		
P	Premature beat followed by compensatory pause	Absent
QRS		Marked irregular

Treatment

- Treatment of the underlying cause.
- Sedation may be needed
- Antiarrhythmic drugs are indicated if premature beats were:
 1. Multiple.
 2. Multifocal.
 3. Occurring in runs.
 4. R on T phenomenon.
 5. Causing symptoms.
 6. In association with acute myocardial infarction.

Amiodarone, β blocker, Ca channel blocker or quinidine are used except in myocardial infarction, In which lidocaine is indicated.

Wolf-Parkinson-White (WPW) syndrome (Pre-excitation)

Def:- It is an accessory pathway between atrium & ventricle (bypass the AVN).

C/P:-

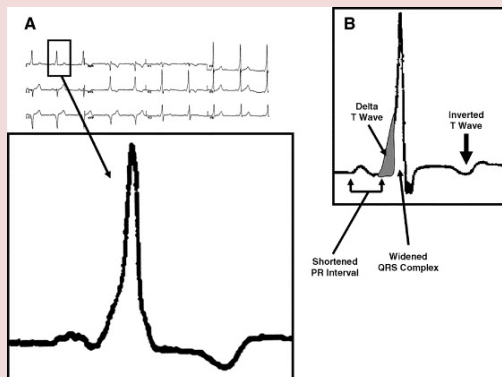
- More in men - associated with MVP, thyrotoxicosis or cardiomyopathy
- Usually asymptomatic
- Or any supraventricular tachycardia:- (S. tachy-PSVT-AF)

ECG:- Short P-R interval - wide QRS complex - delta wave

Treatment :

Medical :- Amiodarone , β blocker (Digitalis is contraindicated).

Surgery:- Ablation.



Sick sinus syndrome (SSS):- rare dysfunction of SA node
may be presented with:-

- Sinus bradycardia
- Sinus arrest (junctional rhythm)
- Atrial fibrillation (slow form)
- Ectopic atrial tachycardia -- prolonged asystolic period

Sinus bradycardia

Definition

It is a condition in which the SAN discharges at a slow rate $<60/\text{min}$ & regular rhythm.

Aetiology (3Ps)

Physiological: During sleep & in athletes.

Pathological: Myxoedma, obstructive jaundice, post-viral, increased ICT, excessive vagal stimulation & sick sinus syndrome (????).

Pharmacological: B-blockers, digitalis, verapamil or reserpine.

Clinical Picture

Symptoms

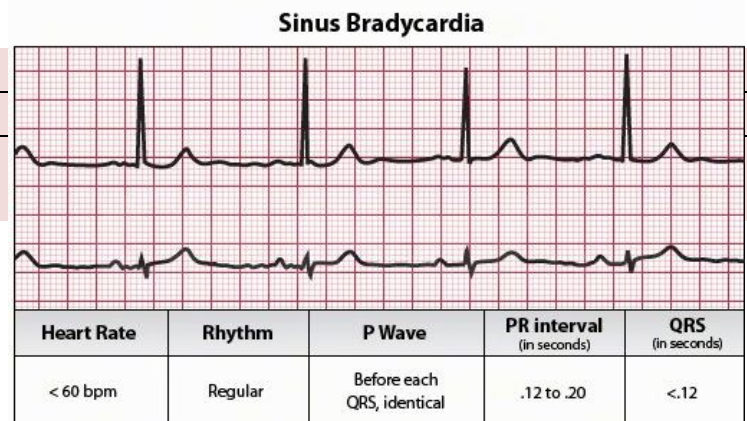
- May be asymptomatic - Palpitation: rare
- Symptoms of low CO may occur in severe cases.
- May precipitate heart failure in susceptible patients.

Signs

Pulse	Rhythm	Regular
	Rate	50-60/min & may be less
	Response to Exercise:	gradual acceleration then gradual returns to the HR
	Res. sinus rhythm	Positive
Neck V	Rhythm	Regular
	Rate	50-60/min & may be less
	Wave	Normal
Auscultation	S1	Normal S1 (may be muffled).

Investigation:-

ECG	Rhythm	Regular
	Rate	50 - 60/min & may be less
	Waves	Each P followed by a QRS



Treatment

1. Treatment of the underlying causes.
2. Atropine is given in symptomatic cases.
3. Presence of marked symptoms may require pacing.

Junctional (nodal) rhythm

Definition

It is a condition in which the (AVN) becomes the pacemaker of the heart. The impulses spread to activate the atria & ventricles simultaneously.

Aetiology:

Cardiac causes:

1. Ischaemic heart disease especially myocardial infarction.
2. Myocarditis & cardiomyopathies.
3. Rheumatic & congenital heart diseases.

It may occur in apparently normal individuals.

Clinical Picture

Symptoms

- May be asymptomatic
- Palpitation : rare.
- Symptoms of low CO may occur in severe cases.
- May precipitate heart failure in susceptible patients.

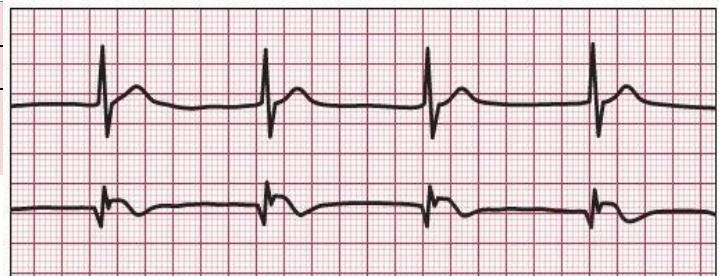
Signs

Pulse	Rhythm	Regular
	Rate	40-50/min & may be less
	Response to Exercise:	reversion to initial HR may occur in some patients
	Res. sinus rhythm	Negative
Neck V	Rhythm	Regular
	Rate	40-50/min & may be less
	Wave	Regular cannon waves with the same rate of pulse.
Auscultation	S1	Regular cannon sound.

Investigations

ECG	Rhythm	Regular
	Rate	40 - 50/min & may be less
	Waves	P wave is <i>inverted</i> or <i>absent</i> QRS normal morphology

Junctional Rhythm



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
40-60 bpm	Regular	Inverted, absent or after QRS	<.12	<.12

Treatment

1. Treatment of the underlying causes.
2. Atropine is given in symptomatic cases.
3. Presence of marked symptoms may require pacing.

Heart block (HB)

A-V Block

- 1st Grade: P-R interval >0.22 sec.
- 2nd Grade:
 - ✓ Mobitz type one: **Wenckebach** phenomenon.
 - ✓ Mobitz type two.
- 3rd Grade: Complete heart block.

Bundle block

- Right bundle branch block (RBBB).
- Left bundle branch block (LBBB).
- Left anterior hemiblock (LAHB).
- Left posterior hemiblock (LPHB).

ATRIOVENTRICULAR BLOCK

Definition:- Delayed conductivity through AV node.

Aetiology

Cardiac causes

1. Ischaemic heart disease especially myocardial infarction.
2. Myocarditis & cardiomyopathies.
3. Rheumatic & congenital heart diseases.

General causes

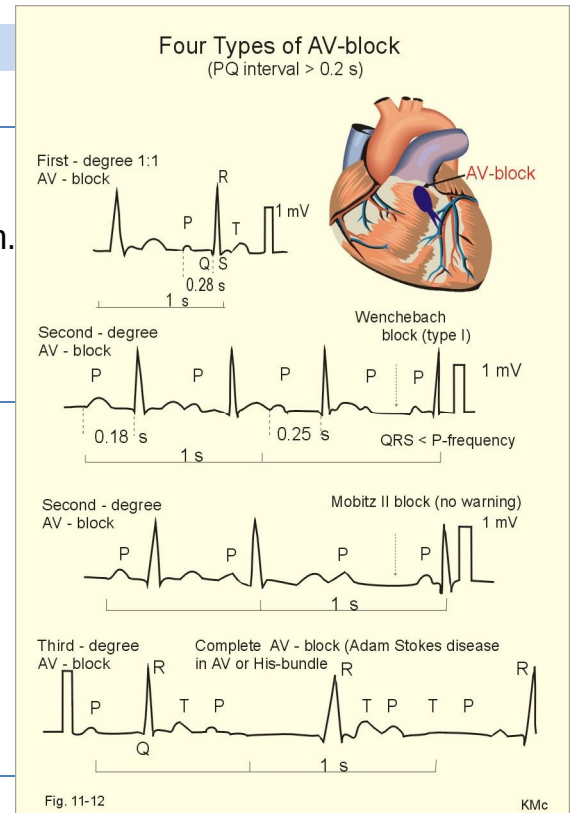
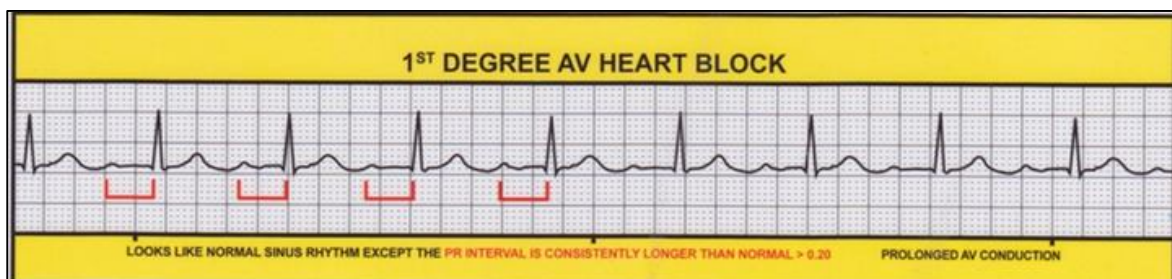
1. Simple causes (stress, sadness, smoking, coffee and tea)
2. Electrolyte disturbances: e.g., Hyperkalaemia)

Specific causes

1. Drugs e.g. B-blockers
2. Fibrosis & calcification of the AV node & bundle (as in calcific AS)

First Grade A-V Block

- It is **prolongation** of **P-R** interval more than **0.22 sec**.
- Clinically **asymptomatic** but weak S_1 may be detected.
- Treatment: treatment of the underlying cause & **atropine** may be given.



Second Grade A-V Block

There are two types:

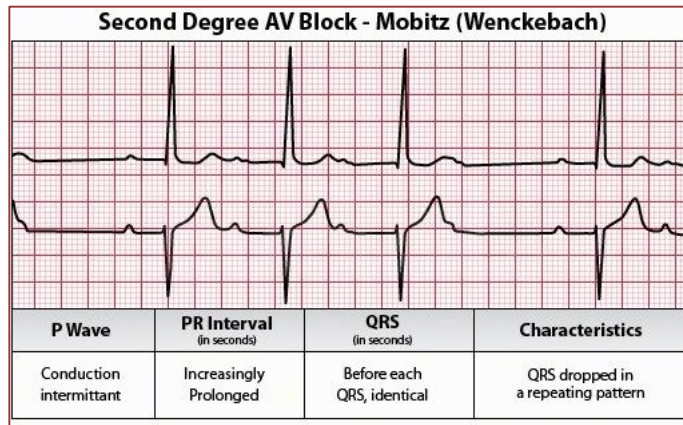
1. Mobitz type 1 (Wenckebach phenomenon):

Definition

- Progressive prolongation of the P-R interval.
- Followed by dropped QRS complex.
- The sequence is repeated.

Clinically:- There is irregular pulse & variable S₁.

Treatment:- Treatment of the underlying cause & **atropine** may be given.



2. Mobitz type 2:

- The AVN transmits one impulse for each 2, 3, 4 or more atrial activities.
- The block may be fixed (e.g. 2:1, 3:1) or variable.

Clinical Picture

Symptoms

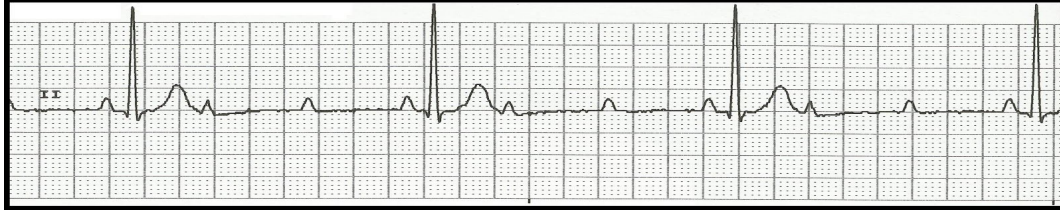
- May be **asymptomatic**. - Palpitation: **rare**.
- Symptoms of low CO may occur in severe cases.
- May precipitate **heart failure** in **susceptible patients**.
- **Adams-Stokes attack** may occur during transmission from partial to complete heart block.

Signs

Pulse	Rhythm	may be regular or irregular (Variable conductivity)
	Rate	40-50/min & may be less
	Response to	Exercise: increase in mathematical fashion then returns to its original level by the same fashion
	Res. sinus rhythm	positive (Hardly noticed)
Neck V	Rhythm	regular or irregular
	Rate	30-50/min & may be less
	Wave	multiple A waves before each V
Auscultation	S1	Normal S1 (may be muffled).

Investigations

ECG	Rhythm	regular or irregular
	Rate	40 - 30/min
	Waves	Multiple Ps (2, 3 or more P waves) between each successive QRS



Treatment

1. Treatment of the underlying causes.
2. Parasympatholytics: Atropin 0.5 - 2.
3. Sympathomimetics: Isoprenaline 0.5-5 ug /min IV.
4. Presence of marked symptoms may require pacing.
5. Treatment of heart failure if present.

Third grade heart block (complete heart block)

Definition

There is complete absence of A-V conduction, so the atria will be controlled by the SAN while the ventricles are controlled by an idioventricular rhythm (A-V dissociation).

Clinical Picture

Symptoms

1. May be asymptomatic. - Palpitation: **rare**.
2. Symptoms of **low CO** may occur in severe cases.
3. May precipitate **heart failure** in **susceptible** patients.
4. **Adams-Stokes attack** may occur during transmission from one idioventricular focus to another (During the attack, there is syncope. Absent pulse & low or absent BP and cyanosis. If the attack is prolonged, convulsions, coma & death may occur.)

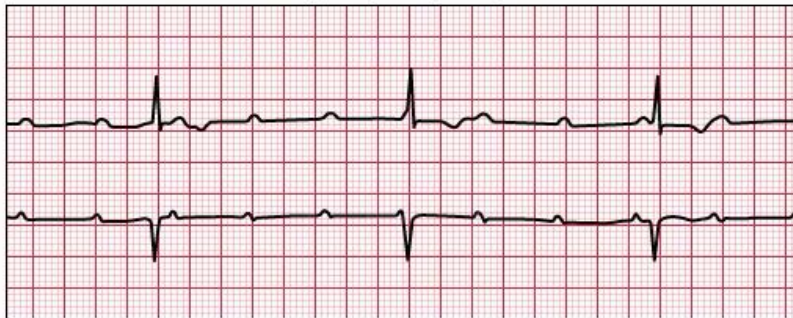
Signs

Pulse	Rhythm	Regular
	Rate	25-40/min & may be less
	Response to Exercise:	Better to be avoided or no effect
	Res. sinus rhythm	Negative
Neck V	Rhythm	regular
	Wave	with occasional cannon A waves of normal rate (60-100/min)
Auscultation	S1	Normal S1 (may be muffled) with occasional cannon.

Investigations

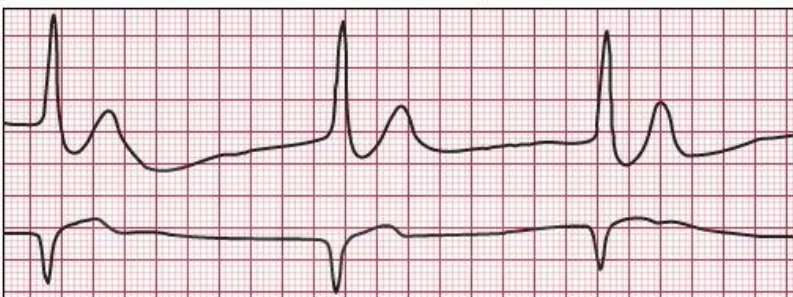
ECG	Rhythm	Regular & slow
	Rate	25-40/min
	Waves	The QRS complexes are widened, bizarre or may be normal . The P waves are of normal rate (60 - 100/min) & morphology and may be superimposed on QRS (A - V dissociation).

Third Degree (complete) AV Block



P Wave	PR Interval (in seconds)	QRS (in seconds)	Characteristics
Normal but not related to QRS	None	N/A	No relationship between P&RS

Idioventricular Rhythms



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
20-40	Regular	Absent or not related	N/A	$\geq .12$

Treatment

1. Treatment of the underlying causes.
2. Parasympatholytics: Atropin 0.5 - 2
3. Sympathomimetics: Isoprenaline 0.5 - 5 ug / min IV.
4. Presence of marked symptoms may require pacing.
5. Treatment of heart failure if present.

Antiarrhythmic agents (Vaughan Williams classifications)

Class	Examples	Clinical uses
I (Na⁺) blocker a	- Quinidine - Disopyramide	- Broad spectrum - Procainamide in <u>WPW</u>
I-b	- Lidocaine - Mexiletine	- Ventricular tachycardia (with risk of V. asystole)
I-c	- Encainide - Propafenone	- Broad spectrum (2nd line) - Cl:- post-MI.
II Beta-blockers	- Propranolol - Timolol - Atenolol	- Tachyarrhythmias - decrease <u>MI</u> mortality
III K-blockers Prolonging repolarization	- Amiodarone - Ibutilide - Dronedarone	Broad spectrum WPW (sotalol)
IV Ca-blockers	- Verapamil - Diltiazem	Supraventricular arrhythmias
V Q; Direct nodal inhibition	- Adenosine - Digoxin - Magnesium Sulfate	Supraventricular arrhythmias (mainly in HF) Cl:- V. arrhythmias.

Class one A

	Effect	Indication	Side effects
Quinidine	- Decrease conduction and excitability. - Slow S.A. node. - Vasodilation.	- PSVT. - AF. WPW. V. tachycardia.	- Hypotension & tachycardia. - Depresses vagal tone. Cinchonism (GITD, tinnitus, headache, auditory and visual disturbances and vertigo). - Hypersensitivity.

Class one B

	Effect	Indication	Side effects
Lidocaine	Depression of rapidly depolarizing tissue.	V. tachycardia. - VF - W.P.W.	- C.N.S. effects: - Muscle twitching - Mentality:- psychosis and seizures. - CVS:- suppression

Class two :- B-Blocker (see IHDs and HTN)

Class three

	Effect	Indication	Side effects	Contraindications
Amiodarone has a low incidence of pro-arrhythmic effects	Reduce heart rate.	- Life-threatening ventricular arrhythmias. - supraventricular arrhythmias	- Pulmonary toxicity and fibrosis. - Liver disease. - C.N.S. symptoms. - Hypothyroidism or hyperthyroidism. - Cutaneous photosensitivity and blue-gray discoloration. - Peripheral neuropathy. - Increased levels of LDL.	2nd or 3rd degree heart block. - cardiac shock

Class four *Ca-Blocker (see IHDs and HTN)***Miscellaneous**

	Effect	Indication	Side effects	Contraindications
Adenosine	Decrease AV nodal conduction.	- PSVT. (Used acute)	- Flushing. - Shortness of breath. - Atrioventricular block. - Transient AF. - Headache. - GI upset. - Hypotension. - Paresthesias.	- Heart block. SSS.

Sudden Cardiac Death

Definition

Sudden cardiac death is unexpected natural death from cardiac causes within less than two weeks of recent illness.

Causes

1. Myocardial infarction & ischaemia.
2. Ventricular tachyarrhythmias especially VF.
3. Heart block: Mobitz type II & 3rd grade (**Adam's stock attack**).
4. AS & hypertrophic cardiomyopathy.
5. Fallot's tetralogy.
6. Mechanical causes:
 - Obstruction of blood flow: massive pulmonary embolism ball & valve embolus.
 - Cardiac tamponade.
 - Rupture of heart or aorta.

Clinical presentation

1. **Cardiac**: absent pulse & heart sounds.
2. **Chest**: absent respiration & cyanosis.
3. **C.N.S.**: loss of consciousness, convulsions, brain damage & death.

Treatment

1. Cardiopulmonary resuscitation (CPR):

- A. **A**irway: Removal of foreign materials-Suction - Hyperextension of the neck.
- B. **B**reathing: Mouth to mouth breathing – Mask - Intubations and ventilation.
- C. **C**irculation: Cardiac massage.

2. Further dealing according to ECG pattern:

A. Ventricular tachyarrhythmia VF:

- Defibrillation (300-400 joules) → if no response lidocaine → if no response defibrillation → if no response bretylium → if no response defibrillation.
- (**Response**: VF changes to V. tac) then treatment of V. tac should be admitted.

B. Asystole:

- **Adrenaline** 5-10ml (1 : 10,000) intracardiac or IV (every 5 min).

C. Heart block or severe bradycardia:

- **Atropine** 0.5mg IV (every 10 min up to 5 mg)
- **Isoprenaline** 2-20 mcg/min IV infusion.
- **Cardiac pacing**.

D. Electromechanical dissociation:

- **Intracardiac calcium** 5 ml 10% solution (repeated every 10 min).
- Correction of **acid-base** and **electrolyte**.

3. Treatment of the cause. E.g. Ischemia.

Aortic aneurysm

1. Atherosclerosis (the most common cause).
2. Congenital.
3. Mycotic.
4. Syphilitic.
5. Traumatic.

Site:

- Ascending aorta.
- Arch of aorta.
- Descending thoracic or abdominal aorta.

Clinical Picture:

A. Aneurysm of the ascending aorta:

It gives rise to marked signs & few symptoms:

Symptoms

1. Chest pain may occur.
2. Huge aneurysm:- mediastinal syndrome.
3. Thrombo-embolism.
4. Rupture:- fatal haemorrhage.

Signs:

Precordial examination.

- Over second right intercostals space
 1. Systolic expansile pulsations.
 2. Palpable second sound & systolic thrill.
 3. Dullness over the second right intercostals space.
 4. Left ventricular enlargement may be detected (associated AR).

Auscultation:

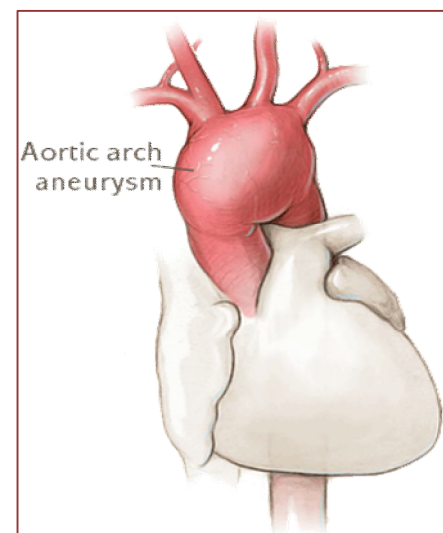
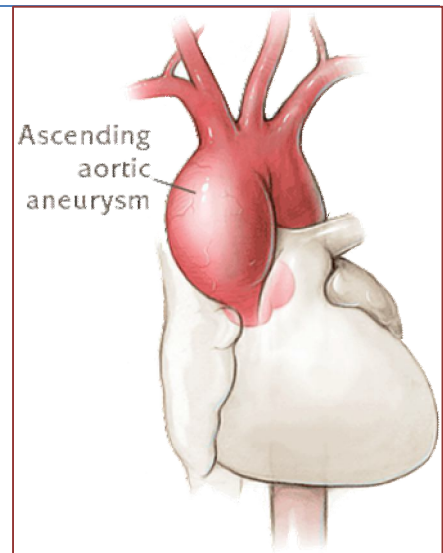
- Over the aortic area reveals:
 - ✓ S2 Loud ringing.
 - ✓ Early diastolic murmur (aortic incompetence).
 - ✓ Ejection systolic murmur (relative aortic stenosis).

B. Aneurysm of the arch of the aorta:

Marked symptoms & few signs:

Symptoms:

1. Chest pain may occur.
2. Mediastinal syndrome.
3. Thrombo-embolism.
4. Rupture of the aneurysm leading to fatal hemorrhage (through the trachea, esophagus, pericardium, pleura, mediastinum or chest wall).



Signs:

1. Systolic pulsations in the suprasternal notch, left infraclavicular region & second left intercostals space.
2. **Tracheal tug**: downward pull on the cricoids cartilage synchronous with the heart beats due to pressure on the bronchi.

Investigations

1. Chest X-ray & Screen:

- Dilatation of the aorta (fusiform or saccular).
- Calcification in the wall may be seen.
- Pulsations of the aneurysm under fluoroscopy.

2. Echocardiography:**3. CT scan & MRI****4. Aortography:** diagnostic**5. Investigations for the cause:** e.g. serological test for syphilis.**Treatment**

1. Avoidance of **exertion**.
2. Treatment of the **cause** if possible.
3. **B-blockers** may decrease the rate of enlargement of the aneurysm.
4. **Surgical** treatment: resection of the aneurysm & replacement with a prosthetic graft is indicated in some patients.

Aortic dissection

1. Hypertension.
2. Cystic medial necrosis & Marfan's syndrome.
3. Pregnancy.
4. Aortic stenosis & coarctation.
5. Trauma.

Classification

- Type one: affecting ascending, arch & descending aorta.
- Type two: affecting ascending aorta only.
- Type three: affecting descending aorta.

Clinical picture

1. Acute severe chest pain which may radiate to interscapular area.
2. Pain may in the neck & jaw (if the aortic arch is involved).
3. Nausea, vomiting & excessive sweating.
4. Acute aortic regurgitation.
5. Rupture in pericardium leading to haemopericardium.
6. Involvement of branches of aorta:
 - a. Carotid arteries: neurological manifestations & unequal carotid pulsations.
 - b. Subclavian arteries: unequal pulse & **low BP** in upper limbs.
 - c. Coronary arteries: ischaemia & infarction.

Investigations

1. Chest X-ray & Screen

- Widening of superior mediastinum.

2. Echocardiography

3. CT scan & MRI

4. Aortography: diagnostic

5. Investigations for the cause: e.g. serological tests for syphilis.

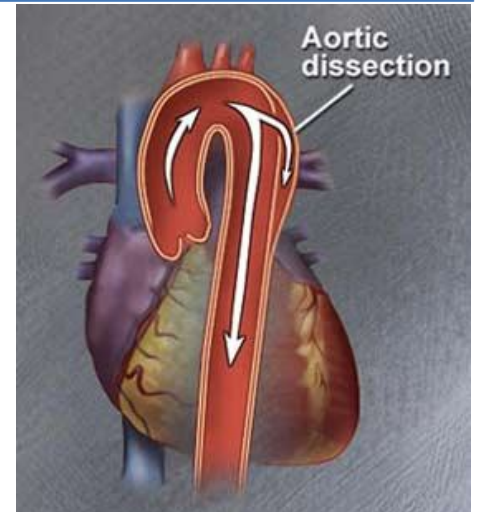
Treatment

Medical treatment:

- **Analgesia.**
- IV infusion of **Na nitroprusside** to reduce the systolic BP to **100 - 120** mm Hg.
- **Propranolol** to keep the heart rate in the range of 60 / min.

Surgical treatment:

- Resection of the affected portion of aorta & replacement with a prosthetic graft.



Cardiogenic shock

Definition

Decreased cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume.

Haemodynamic criteria for cardiogenic shock are

1. Sustained hypotension (systolic blood pressure $< 90 \text{ mm Hg}$ for at least **30 min**).
2. A reduced cardiac index ($< 2.2 \text{ L / min / m}^2$).
3. Elevated pulmonary capillary occlusion pressure ($> 15 \text{ mm Hg}$).

Causes

A. Cardiac causes (causes of rapid reduction of COP)

1. Pump failure: **Causes of LVF**.
2. OUT flow obstruction: as **sever AS** or **S. hypertension**.
3. Valvular dysfunction e.g. **acute mitral regurgitation**.
4. Cardiac arrhythmias **Ventricular tachyarrhythmias**.

B. Extra-cardiac causes

1. Global hypoxemia.
2. Myocardial depressant drugs (e.g., B-blockers, Ca blockers).
3. Respiratory acidosis.
4. Pulmonary vascular disease (e.g., pulmonary hypertension)
5. Acute respiratory distress syndrome.

Clinical presentation

Symptoms Low CO.

Signs

General signs

- Cyanotic, cool skin and mottled extremities.
- Pulses: rapid and faint.
- Jugular venous distention.

Local signs

- Heart sounds usually distant, and both third and fourth heart sounds may be present.
- A systolic murmur is generally heard in patients with acute MR or VS rupture.

Investigations

- as MI

Treatment

- **Vasopressors / inotropic agents**: (Dopamine – Dobutamine).
- **Vasodilators** : as, Nitroglycerin.
- **Analgesics** Morphine sulfate.
- **Diuretics**: as, Frusemide.

Myocarditis

Definition :-inflammation of myocardium

Aetiology (4Is)

Infections	• Viral (adenovirus, parvovirus , HIV, enterovirus, rubella , polio , CMV) • Protozoan (Chagas disease and Toxoplasma) • Bacterial (Brucella, diphtheriae, gonococcus, H.influenzae, Rickettsia) • Fungal (Aspergillus) • Parasitic (ascaris, schistosoma, Taeniasolium, Trichinellaspinalis) <i>Bacterial myocarditis is rare in patients without immunodeficiency</i>
Iatrogenic	Drugs (ethanol and some forms of chemotherapy)
Immunological	• Allergic drug reaction (acetazolamide) • Rejection after a heart transplant • Collagen (scleroderma, SLE, sarcoidosis, systemic vasculitis such as Churg-Strauss syndrome, and Wegener's granulomatosis) • Toxins (arsenic, toxic shock syndrome or snake venom)
Irradiation	(Physical agents) :- Electric shock, hyperpyrexia

Clinical picture:-

- features of the cause :- e.g. viral infection
- Chest pain
- Congestive heart failure
- Palpitations (due to arrhythmias)
- Sudden death (in 20% of cases)
- Myocarditis is often associated with pericarditis.

Investigations:-

ECG:-	- Tachycardia - Any form of arrhythmia - Non specific changes - Features of complications:- IHDs or pericarditis changes
Echo & doppler	cardiomegaly and contractile dysfunction
MRI	Diagnostic
Myocardial biopsy:- the most diagnostic but invasive	
Investigation for detection of the cause e.g. serology of viruses	

Treatment (3Ss)

- 1- Symptomatic treatment:- analgesics and antipyretics
- 2- Supportive :-
 - anti heart failure
 - control of arrhythmia
- 2- Steroids and immunosuppressive drugs

Causes of cardiac murmurs

There are two types of heart murmurs:

- Innocent murmurs.
- Abnormal murmurs.

Innocent heart murmurs A person with an innocent murmur has a normal heart.

- This type of heart murmur is common in newborns and children.
- An innocent murmur can occur when blood flows more rapidly through the heart:
 - Physical activity or exercise.
 - Pregnancy.
 - Fever.
 - Changes in your heart's structure, such as changes from heart surgery
 - anemia
 - hyperthyroidism

Innocent heart murmurs may disappear over time, or they may last your entire life without ever causing further health problems.

Abnormal heart murmurs

An abnormal heart murmur is more serious.

In children, abnormal murmurs are usually caused by congenital heart disease.

In adults, abnormal murmurs are most often due to acquired heart valve problems.

DD:- Diastolic Murmurs

- I. Aortic regurgitation**
- II. Pulmonary valve regurgitation (Functional)**
- III. Mitral rumble**
 - A. Obstruction to flow
 - 1. Mitral stenosis (rheumatic, congenital)
 - 2. Left atrial myxoma
 - B. Increased flow
 - 1. Mitral regurgitation
 - 2. Ventricular septal defect
 - 3. Patent ductus arteriosus
- IV. Tricuspid rumble**
 - A. Obstruction to flow
 - 1. Tricuspid stenosis (rheumatic, Ebstein's anomaly, carcinoid)
 - 2. Right atrial myxoma
 - B. Increased flow
 - 1. Atrial septal defect
 - 2. Tricuspid regurgitation

Systolic Murmurs

I. Ejection murmurs

A. Functional

1. Flow murmur from the root of the pulmonary artery
2. Murmur associated with high COP
3. Flow murmurs associated AR - PR

B. Organic

1. Valvular AS
2. Aortic sclerosis
3. Subvalvular AS (web or tunnel)
4. Supravalvular AS
5. Hypertrophic obstructive cardiomyopathy
6. Pulmonary valvular stenosis
7. Pulmonary infundibular stenosis
8. ASD
9. Tetralogy of Fallot

II. Regurgitant murmurs

1. MR
 - a. Rheumatic
 - b. Papillary muscle dysfunction
 - c. Mitral valve prolapse
 - d. Acute
2. TR (*Functional - organic*)
3. VSD

III. Extracardiac sounds simulating systolic heart murmurs

- A. Subclavian (supraclavicular/brachiocephalic) murmur
- B. Internal mammary soufflé
- C. Carotid artery bruits
- D. Coarctation of the aorta**
- E. Murmurs emanating from a dilated aortic or pulmonary artery root
- F. **Patent ductus arteriosus** with pulmonary hypertension